Anomalies of Infants and Children

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Dedicated to Addie Mayer and Lottie Swanker, Our Wives

FOREWORD

Anomalies of Infants and Children is written primarily for the student and general practitioner I believe it will also be of aid to the obstetrician and pediatrician

The book is designed as a quick reference It helps those who are interested in this subject to discuss intelligently with the parents and others concerned the anatomical anomaly from which the patient is suffering, the resulting physiologic disturbance the rationale of treatment, and the accepted time for repair

The subject of anomalies of infants and children, in the opinion of many writers has never been properly studied or correlated Until a few years ago these anatomical defects fell into the hands of the general surgeon who perhaps had little if any experience in their repair The general surgeon was fortunate who when called to treat congenital or acquired anatomical defects of the palate superior maxilla, or inferior maxilla, could consult with or call to his aid a maxillofacial or plastic surgeon

Plastic surgery may be defined as that specialty which embraces reconstruction of anomalies and anatomical derangements following injury or disease—in fact all operative measures undertaken to improve the appearance and to restore function of the human body The authors' training has qualified them in the field of maxillofacial surgery after basic training in general surgery Their experience in plastic surgery has been extensive, not only in civilian practice but also in wartime surgery

Dr William J Mayo once wrote, "Every human being has the divine right to look human One of the compensations of the Great War was the development of plastic surgery of the face, a new special field in surgery which has given astonishing results"

To learn technique is easy, but to acquire judgment is a different matter Both these attributes are possessed by the authors Plastic surgery is not for the impatient surgeon or patient Patients and their relatives should be made to understand the complication of the congenital or acquired deformity involved, the numerous operations that may be necessary, and the length of time required

Some might be tempted to say that this book describes nothing new or nothing that has not been described by previous writers, this is true, but the present authors are the first to correlate the available information in one volume It was the novelist Charles Reade who, when accused of drawing to an undue extent on previous writers, said, "I have milked three hundred cows but the cheese that I made was my own"

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PREFACE

Throughout their years of experience in the observation and treatment of anomalies in infants and children, the authors have been impressed by an apparent lack of general familiarity with the causes and dangers of many anomalies the prognoses for their correction and the optimum time for the initiation of therapy

This is the more surprising in view of the fact that so much well-documented scientific knowledge and experience in this field has been recorded Unfortunately such information is widely scattered and appears in isolated papers or in brief incidental chapters in books devoted to a single specialty such as orthopedics pediatrics or urology

Until now it has been necessary therefore to refer to many sources in order to locate references to a specific anomaly This book is an attempt to place in a single volume our present knowledge of the more common types of congenital and acquired abnormalities

The chapters have been organized for simple and rapid reference Their subjects include the role of genetics in the

production of congenital malformations, the increasing incidence of the acquired deformities, and cancer in infants and children

Because not only the patient but also the family—especially the mother—may often suffer psychological trauma in connection with anomalies, considerable attention is given to facts which can be used to explain congenital anomalies as a natural hazard of conception and birth rather than a consequence of prenatal influences In each case the authors indicate what can and should be done about the anomaly, when such steps ought to be initiated, and what final outcome can reasonably be expected

The authors call attention to the danger of radiation for the removal of benign skin lesions and have devoted an entire chapter to the management of burns They also stress the critical importance of initial treatment of a burn patient and the avoidance of dressings and medication which may complicate later professional care With these exceptions, little reference has been made to the details of treatment of anomalies, in recognition of the fact that many medical specialties are involved in such treatment

It is the authors' hope that this volume will prove to be a simple and ready reference and will make more general the knowledge of a subject of very pressing importance to thousands of young patients, their parents, their families, and their friends

Grateful acknowledgment is made to the many who helped and advised in the compilation of this book in particular to Majorie Foley, our photographer, to Stanley Q Schwartz and Masayuki M Nakamura of the Medical Illustrations Department, Veterans Administration Hospital, East Orange, New Jersey, and to David Q Hammond, Vice President of Fairleigh Dickinson University

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CHAPTER 1

Introduction

Although a certain amount of surgery has been performed in the young for many years it is only in the last couple of decades that extensive surgery has been carried out in the young as well as in the very old Surgical horizons have been extended in every direction Much of this advance can be attributed to the marked improvements in anesthesiology The development of many new anesthetic agents as well as the perfection of additional techniques in the use of the older agents offers a wide enough choice for the anesthetic to be fitted to the patient rather than the patient to it The present growth of anesthesiology as a medical specialty and the dependence on it by surgeons stimulate better training of anesthetists

The progress of surgery has kept pace with anesthesiology The major strides have occurred in the physiologic aspect of surgery The electrolyte and fluid balance features of preoperative and postoperative treatment have been recognized and embodied in the general knowledge of surgery There is continual improvement in various techniques As new information is uncovered by research better procedures are de-

to early surgery The nose continues to grow until about the sixteenth year If surgical trauma interrupts the normal growth some degree of saddlenose or other deformity may occur Small nasal corrections particularly those of the nasal tip or other soft tissue are permissible if the deformity warrants it However correction of the nose may not be lasting as the eventual shape is unpredictable even when growth centers are not involved Nasal corrections in youth are usually limited to the secondary nasal tip deformity associated with cleft lip and palate, and traumatic defects that demand immediate repair such as dislocation of the parts resulting in obstruction

Very early surgery especially in the treatment of palatal defects is imperiled by so many anesthetic or physiologic problems that it is usually not attempted Many surgeons feel that the period from the twelfth to the eighteenth month is the optimal time for corrective surgery of the palate Others prefer to defer surgery until the maxillary arch has had more time to develop unhindered This ensures more normal dentition In treatment of cleft palate, one of the chief aims is normal speech This is rarely achieved by early cleft palate surgery without an effective speech training program for the child as soon as possible after closure A dental prosthesis or an obturator helps in swallowing and permits the patient to form good speech habits The normal growth of the maxillary arch is not restricted and good dentition is encouraged At a later date about the fifth year or later surgical correction can be done more safely and with a chance of anatomic perfection

The reconstruction of the cleft lip need not await the cleft palate closure As no skeletal interference is present in the lip proper the closure of the upper lip should be performed soon after the child has regained its birth weight If other pertinent factors are favorable a protruding premaxilla of a double cleft can be repositioned at this time in order to effect a closure. Complete or nearly complete cleft of the upper lip should be repaired within the first three months

veloped. Procedural controversies are resolved by common interest and combined effort of surgeon and anesthetist.

The impact of an anomaly, especially a visible one, is fully realized only by the one afflicted or by those close to him. Others of us feel compassion toward such an unfortunate person but we can never, of course, measure his profound psychologic depression, which is the same whether the deformity is congenital or acquired.

A conspicuous anomaly initiates a marked personality change in most adult victims. In the infant and the child, the effect is first of all on the immediate family, particularly the mother, until the child enters the preschool age, when he meets new friends and playmates, some of whom may be unkindly critical. At this time the child realizes how different he is from other children. To protect himself from unkind nicknames and remarks, his tendency is to avoid others altogether, and thus the psychologic harm begins.

This emphasizes the importance of early surgery. For the child's benefit, surgery should be performed well before he has reached the immediate preschool age. For the family's benefit (or peace of mind), surgical correction should be made as soon as possible after birth.

In many anomalies of the congenital type, the correction can be made shortly after birth. The only deterring factors are possible interference with the growth centers and the patient's ability to withstand surgery, including anesthesia. If the growth centers are traumatized by the procedure, a cessation of growth may result in a new abnormality superimposed on the original one. In fact, the postoperative defect may be worse than the original anomaly, which may have involved only the soft tissues. The concern for the growth centers is especially important in skeletal anomalies, more so than in those affecting soft tissue. This possibility usually precludes the use of radiation of any type in infants and children.

Among the facial structures, the nose is the least amenable

CHAPTER 2

General Aspects of Pediatric Surgery

Surgery in the infant or child is a specialized field not merely a scaled-down form of adult surgery Besides the variations of immature anatomy and physiology there are specific indications and special skills with which the pediatric surgeon must be familiar Patients in this age group show a tolerance for surgery in direct proportion to the surgeon s ability and plan ning They particularly lack tolerance for loss of blood, and their sensitivity to exposure and trauma requires short opera tive procedure gentle tissue manipulation and expert adminis tration of anesthesia

HISTORY

Much can be learned of the patient and his condition by careful history taking including the past history as well as the illness or anomaly currently under consideration In recording data, it is best to express age in hours for the newborn infant

In general, other deformities, congenital or acquired, should be eradicated by surgery as early as possible. Some birthmarks, it must be remembered, regress or disappear Therefore surgery may be delayed indefinitely, as these defects are of a cosmetic nature and seldom involve function It is well to inform parents of this

Many pediatricians measure an infant's health by its gain in weight When the infant has oriented itself to its new environment well enough to regain the ounces which are normally lost shortly after birth, it is able to withstand the shock of minimal surgery With acquired defects, the time to operate depends on the degree of the trauma rather than on the general good health of the patient It is best to combine the reconstructive surgery with first-aid treatment, as frequently a secondary procedure can be eliminated in this way The patient's contours as they will be in adulthood—particularly the face—must be carefully considered when reconstructive work is performed in youth If skeletal corrections are necessary, it is advisable to defer any attempt at final reconstruction until after puberty

An axiom to follow concerning all deformities is that when restoration of function is imperative, cosmesis is secondary The surgical plans should, however, consider both the functional and the cosmetic results

It has been the authors' aim to compile material that will be of service to the general practitioner, the dentist, and the pediatrician in acquainting their patients or patients' relatives with the best time for and the possibilities of reconstructive surgery Therefore, little space has been spent on techniques and operative procedures Just enough of the method is presented to equip the reader with the essential means for visualizing the problem Readers who wish to investigate more thoroughly all the steps of the procedures can find them in any of a number of textbooks on general plastic surgery

PHYSICAL EXAMINATION

Although a complete physical examination is desirable it is often advisable to proceed directly to the focal condition and evaluate the findings in the light of the proposed surgery It is well to remember that a trusting child will frequently exhibit unusual fortitude An explanation of what is to be done if the child is old enough to understand and cooperate will often be rewarded by complete cooperation The well advised physician will even tell the child that the examination may be painful but if severe pain must be inflicted sedation or if necessary general anesthesia should be administered

Such objective findings as the temperature pulse respiration and blood pressure must be evaluated according to the child's age The temperature in infants and young children averages slightly higher than in adults Extremes of temperature over a broad range are produced by slight causes in the child However a rise in temperature does not always mean that infection is present. Infants and young children show temperature elevations unaccountably or for trivial causes such as slight gastric upsets colds teething, or mild neurogenic disturbances such as might be induced by environmental changes

The pulse is normally irregular in the infant It ranges from 130 to 140 beats per minute at birth to 110 to 120 in the first and second years and 90 to 100 from the second to the fourth year finally slowing to the level of 75 to 80 at puberty Crying or any trivial excitement may increase the rate 20 or 30 beats per minute even in adults

The respirations of the infant are irregular varying from an average of 25 to 35 per minute up to the first year and gradually diminishing to about 18 to 20 per minute during the fifth year The breathing is usually of the abdominal type until the tenth year after which the costal type predominates

The blood pressure is impossible to determine by the usual

record is about 1600 B C, and Breasted[10] believed that it was an incomplete copy of a much earlier writing, perhaps as old as 3000 B C and possibly representing the teachings of Imhotep

The papyrus classifies head injuries as lacerations, fractures, compound fractures, and compound comminuted fractures, and describes each type carefully It likens a puncture of the cranium, for example, to a hole in the side of a pottery jar With an open, comminuted fracture, the brain may be ruptured· "the membrane enveloping the brain is rent so that it breaks open his fluid in the interior of his head " This is the earliest recorded reference to the meninges and cerebrospinal fluid In a closed comminuted fracture, it was noted, "his eye is askew because of it, on the side of him having that injury which is in his skull " The folds of the brain lying in convolutions are described as "corrugations which form on molten copper " And the pulsations of the brain were recognized as "something throbbing and fluttering under thy fingers like the weak place of an infant's crown before it becomes whole " Head injuries could produce facial distortions, motor defects in the extremities, and loss of speech from a wound in the temporal region The directions for examining a person with a head injury included palpation of the wound, observation of the patient's movements and postures, and his ability to speak Even the examiner's sense of smell was called on

Lacerations were treated with adhesive material and, later, by suture Treatment of fractures was conservative, judging by the skulls of ancient Egypt Fragments of bone were extracted and wounds sutured, but evidence of trephination has been found in a single skull only "Fresh meat" was applied on the first day, thereafter, a dressing of lint saturated with grease and honey was used

Greece

There is some evidence that Greek medicine inherited many of the Egyptian methods Hippocrates's method of treating a dislocated

mandible, for example, is similar to one described in the *Edwin Smith Papyrus* for the same condition.

The surgeon's art was highly esteemed in ancient Greece. Between the time of Homer and that of Hippocrates, a matter of some 600 years, considerable medical lore accumulated on anatomy, physiology, pathology, military medicine, and medical education. Greek civilization emphasized harmonious, healthful living. The great leader and teacher in the healing art was Hippocrates,[13] the son and grandson of physicians, who was born in 460 and died in 370 B.C. Although he is better known for his contribution to the ethics of medical practice, his rational ideas on surgery and medicine are equally great. His clear, concise writings bear witness to a rich clinical experience, keen observation, and clear reasoning. In one of his treatises, which included many examples of fatal outcome, he states "I have written this down deliberately, believing it is valuable to learn of unsuccessful experiments and to know the causes of their failure."

Hippocrates held that no head injury was trifling, since even wounds involving only the integument might prove to be dangerous if neglected. He divided skull injuries into simple fractures or fissures, contusions without fracture or depression, fractures with depression, indentation or a cut in the outer table of bone not necessarily attended with fracture or contusion, and counterfissure or fracture by contrecoup. He advised trephination in the first two types, pointing out that when a fracture could not be made to disappear by scraping, the trephine was to be applied. He thought operation should not be performed in extensive depressions, since such fractures were no more dangerous than other skull injuries of less formidable appearance. When performing trephination, the skull was to be penetrated down to the meninx, and the bone was to be sawed nearly through, leaving the piece of bone to exfoliate. He cautioned against removing bone fragments in depressed fractures. His instruments included the modiolus, later described by Celsus[16] (Fig. 3 *a*), which was not unlike the modern

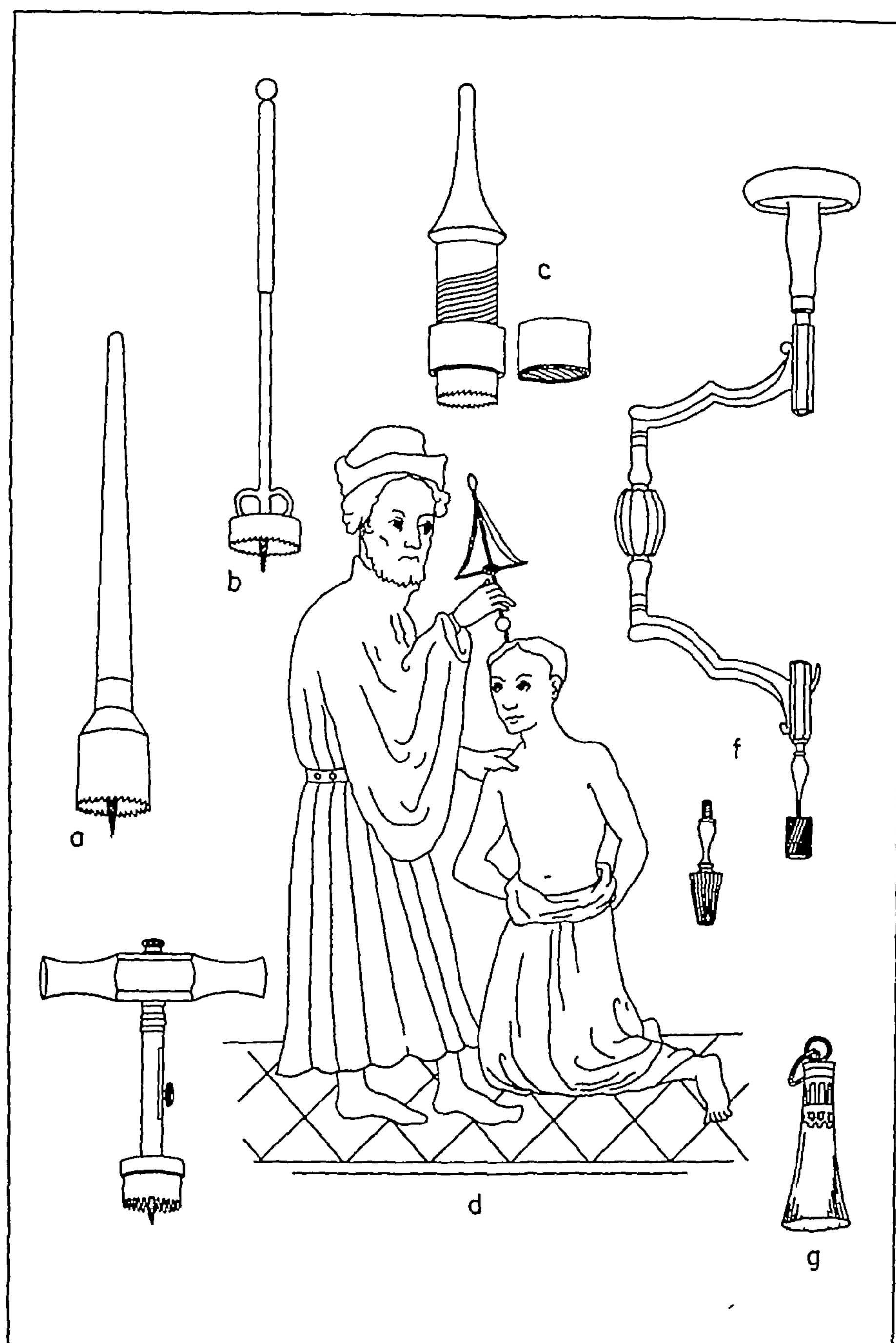


Fig 3 Circular saws of various periods (*a*) Third century modiolus[75] (*b*) Fourteenth century trepan[75] (*c*) Sixteenth century saw[75] (*d*) Trepanation (from a manuscript at Oxford University)[75] (*e*) Late eighteenth century trephine[75] (*f*) Eighteenth century brace and trephine[40] (*g*) Seventeenth century bone brush[40]

trephine. Liquids, cataplasms, and bandages were to be used only for wounds on the forehead or the face.[37]

In the next several centuries, the great schools of anatomy and medicine in Alexandria flourished and declined, and considerable contributions were made to the knowledge of the anatomy of the nervous system. No writings of Herophilus and Erasistratus, founders of the school of anatomy, are extant, and we know about their work only through the writings of Galen,[29] Celsus,[16] and Oribasius.[60] Both dissected the human body and both investigated the nervous system Herophilus differentiated between the cerebrum and cerebellum, and described the torcular, choroid, retina, dura, pia, and the fourth ventricle. He also described the pulmonary artery, which he called the arterial vein He counted the pulse with a water clock, and made a study of its rate and rhythm

Rome (156 B.C.–250 A.D)

Celsus and Galen are the most important medical figures of this period Celsus, a resident of Rome about 30 A.D., was not a physician, but he compiled and translated treatises on medicine, agriculture, and related subjects. His medical works included the first account of the use of the ligature and a description of lithotomy

In his discussion of head injury, he drew freely on the writings of Hippocrates. He recommended that the examiner ascertain whether the patient had vomited bile, experienced dimness of vision, loss of speech, discharge of blood from the nose and ears, and whether he had fallen down at the time of the injury and lain senseless, these symptoms might indicate a skull fracture and the injury was to be regarded as serious. Torpor accompanying mental aberrations, paralysis, or contractures of the tendons pointed to injury of the cerebral membrane and there was little hope for recovery in such cases. He recommended examination with a specillum (sound) that was neither too sharp nor too blunt to ascertain whether or not the bone was fractured. A detectable roughness indicated a fracture, if the bone felt smooth, it was not

broken He warned against mistaking a suture for a fracture, a mistake admitted by Hippocrates If a fracture could not be detected by this method, ink could be poured on the suspected part and the bone scraped, the bone would show a black discoloration were a fissure present He pointed out that the fissure might occur at a distance from the site of the blow, and called this a fracture by repercussion He noted that a blood vessel in the cranial cavity might be torn even though the skull was not fractured, in such cases, the bone would look pale, an observation confirmed by Abernethy[1] many centuries later

The operations for head injuries described by Celsus are probably representative of his period If the injured portion of the bone was not sufficiently exposed, the wound was enlarged and the periosteum was scraped away with a raspatory When the integument was not lacerated, the skull was exposed by an incision consisting of two crossed lines intersecting each other in the form of the letter *X*. Bleeding was stopped by a sponge dipped in vinegar or by compresses and elevation of the head Dressings of wool soaked in vinegar and bandages were recommended for cranial fractures and fissures If the skin began to heal and the fever to subside, this treatment was continued, the fissures often filled with callus in such cases But if fever set in, with disturbed sleep, loss of appetite, and a copious discharge from the wound, operation was necessary In a depressed fracture, a minimum of bone was to be removed, when the edges of bone overlapped, the overlapping part was to be removed with a raspatory In fractures in which the edges were firmly pressed together, a hole was bored a finger's breadth distance away with a wimble or terebra, and a *V*-shaped incision was made to the fissure, with the vertex of the *V* at the hole and the base at the fissure Long fractures might require more than one hole Depressed portions of bone were then carefully removed with suitable forceps (Figs 2-3)

Galen, who lived in Rome from 131 to 201 A D, was a skillful practitioner who accomplished miraculous cures, mainly by the use of polypharmacy He wrote voluminously, and his works form a huge

encyclopedia of knowledge. His work on neurologic anatomy is excellent. He identified the dura, pia mater, corpus callosum, third and fourth ventricles, and the sylvian aqueduct. He also identified seven of the twelve cranial nerves, recognized the sympathetic ganglions, and described the vein which bears his name. Until the time of Harvey, he was the foremost contributor to experimental physiology, and he was the first experimental neurologist: he sectioned the spinal cord, thereby causing paraplegia, he produced aphonia by cutting the recurrent laryngeal nerve, and he gave the first valid explanation of the mechanism of respiration.

For hemorrhage, Galen advised placing the finger lightly on the mouth of the bleeding vessel, and ligating the vessel if necessary — an early observation on the value of the ligature. Nevertheless, despite his greatness, some of his dictums long prevented the advancement of medical knowledge. One of these was his concept that vital spirits in the blood were transformed into animal spirits in the brain and supposedly coursed back and forth through the hollow nerves. His belief that coction (suppuration) was essential to the healing of wounds led to the use of setons and the concept of "laudable pus." This concept, although combated by de Mondeville,[61] Paracelsus,[62] and Paré,[63] was not entirely vanquished until the time of Lister.

Galen classified cranial fractures into those which extended to the diploë and those which penetrated the inner surface of the bone, and he divided the fractures into simple fractures, contusions, and depressions. He listed three types of instruments for operations on the cranium: the cyclisci, the lenticulari, and the raspatori. Comminuted depressions were to be removed entirely, but if fracture lines extended farther, they were not to be followed to their ends. He apparently used the trephine occasionally, but he preferred hollow chisels which he drove with a hammer.

Midthird to Midnineteenth Centuries

The Dark Ages

For almost fourteen centuries after Galen's death, European medicine remained in a static state All through the Dark Ages and into the sixteenth century, European medicine was involved in a vast argument in which everything relating to anatomy, physiology, and disease was referred back to Galen as final authority

A few names emerge from the Dark Ages Oribasius in the fourth century wrote a medical encylopedia This was the main source for Paulus of Aegineta,[64] in the seventh century, his sixth book dealt with surgery He classified skull fractures as fissure, incision, expression, depression, arched fracture, and, in infants, a dent He gave specific directions for the treatment of head injuries Shave the head about the wound and make two incisions intersecting at right angles, like the Greek letter *X*; one of the incisions may be the wound itself Lay bare the bone to be perforated, apply pledgets of oxycrate or a compress of wine and oil if there is bleeding If the bleeding is too heavy, apply a compression bandage and proceed with the skull perforation the following day The patient may be seated or be placed in a reclining position His ears should be stuffed with wool so that he does not hear the noise of the perforator After removing the dressing, assistants retract the four angles of the incision, and loose fragments of bone, if present, are removed The skull is perforated by an abaptiston (an instrument so constructed that it cannot penetrate into the brain) The bone is then broken by chisels and removed piecemeal with the fingers or with a tooth forceps, bone forceps, or nippers Several perforations may be made, and the bone between the perforations removed The dressings applied after exposing the entire area without injuring the dural membrane consist of a linen rag the size of the wound dipped in oil of roses and placed on the dural membrane Then a small ball of wool saturated with the oil and a compress dipped in wine and oil

is used to cover the whole wound. A bandage holds the dressing in place. The dressing should be loosened on the third day and the meninges sprinkled with cephalics.

Islam's principal service to medicine was its preservation of Greek knowledge after the capture of Alexandria by the Mohammedans. Many of the medical writings in Arabic of this period were by men of Persian or Spanish birth, others were by Jews who reached Alexandria after the destruction of Jerusalem. Mohammedan medicine had its start in the fifth century, with the information collected by a persecuted sect of Christians led by the priest Nestorius who had fled from Constantinople. In the following centuries, the works of Hippocrates, Galen, and others were translated into Arabic, and Abulcasis's[2] encyclopedia of thirty books, three of which dealt with surgery, appeared in the eleventh century. Most of the material in it was copied from Paulus of Aegineta. The books on surgery described cautery (the special feature of Arabic surgical technic), methods of extracting arrows and of amputations, and the treatment of wounds, fractures, and dislocations. The treatment of head injuries was almost identical with the methods advised by Galen and Paulus.

The Crusades of the eleventh and twelfth centuries were the principal means in spreading Mohammedan knowledge to Europe. To the school of medicine at Salerno, established in the eleventh century, and the first in Europe, came Jewish physicians from the Near East as teachers, for they were among the few who had any knowledge of ancient learning at that time.[18] Constantinus Africanus[73] and Maimonides[82] (Rabbi Moses ben Maimon), the personal physician of Saladin, flourished in the eleventh and twelfth centuries. The former, supposedly a native of Carthage, spent many years traveling in Arabia, India, and Egypt. He collected copies of the works of the Greek and Arab writers, finally settled down at the Italian monastery of Monte Cassino, and devoted the rest of his life to translating his collection into Latin.

The great universities of Paris, Oxford, and Cambridge were estab-

lished in the first years of the thirteenth century So were the schools of Naples, Bologna, and Montpelier The Church was the dominant force in the schools, as it was in all other departments of life Priests were the physicians, surgery was the province of the barbers, progress in all fields of medicine was at a standstill Among the few names in medicine that come to us from this century are those of Theodoric,[74] Gulielmus de Saliceto[36] (William of Salicet), and Henri de Mondeville [58]

Theodoric treated wounds by a "dry" method because he disagreed with Galen's idea that "coction" or suppuration was essential for wound healing Gulielmus de Saliceto used the knife instead of cautery, and wrote about head injuries De Mondeville believed that the studv of anatomy was a fundamental requisite for the practice of surgery

The outstanding surgeon of the fourteenth century was Guy de Chauliac [38] He was physician to three popes at Avignon, and the author of a treatise on surgery He described the escape of cerebrospinal fluid in skull fractures and noted the effect of increased pressure upon respiration

With the introduction of gunpowder, first used at the Battle of Crécy in 1346, a new type of head injury appeared — the penetrating wound caused by a missile Hieronymus Brunschwig,[15] a barber surgeon of Strasbourg, in his book on surgery published in 1497 advised cauterization of the wound by boiling oil of elder or by the cautery The first work on surgery to appear in the English language was the translation of Brunschwig's treatise He recognized fracture of the inner table of the skull without involvement of the outer table Giovanni de Vigo[32] also described gunshot injuries and their treatment Burns, contusions, and gunpowder poisoning, according to his work, were concomitants of injuries due to bullets, and were therefore difficult to cure His treatment consisted of moist applications and desiccation, and cauterization *with the boiling oil of elder or by the cautery to keep the wound from* putrefying

Many others contributed to knowledge of surgery, medicine, and

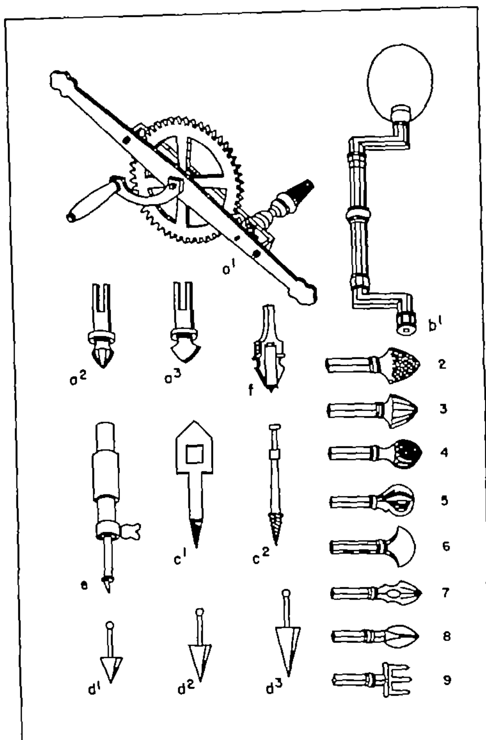


Fig 4 Perforators and drills of various periods. Sixteenth century drills of Narvatio of Antwerp (*a*)[72] and Berengarius (*b*) fifteenth century perforators of Lanfranc (*c–d*)[73] sixteenth century perforators of Paré (*e*)[62] and Berengarius (*f*)[7]

anatomy in the sixteenth century The list of names is long Ambroise Paré,[63] possibly the greatest surgeon of his century, departed radically from the customary treatment of head wounds, he discarded use of cautery and merely dressed the wounds after applying a digestive He introduced ligature instead of cautery for bleeding arteries after amputations His treatise on gunshot wounds included a summary of Vesalius's anatomy, and he turned from dependence on the ancient writings, stating "You will have to render account not to the ancients but to God for your humanity and your skill"

Paracelsus,[62] army surgeon and professor of medicine at the University of Basel, influenced European medicine for over a century, and some of his theories survive in modern homeopathy He argued that suppuration was not necessary for wound healing

Felix Wurtz,[82] a follower of Paracelsus, advised conservative treatment of wounds, believed trephination inadvisable for skull fractures caused by gunshot, and held that complicated instruments should not be used to extract bullets from the cranial cavity He condemned the use of styptics and cautery, except in amputations at the thigh Another German surgeon who raised the standard of surgical practice was Fabricius Hildanus[25]

Joannes Andreas a Cruce[19] taught in Venice, and was the author of a well-known treatise, he employed several types of instruments for trephination, including a brace and drillstock to which circular saws and perforators were fixed with a screw (Fig 2) Narvatio Matthia[60] of Antwerp invented a mechanical instrument operated by a cogwheel for trephination Hieronymus Fabricius ab Aquapendente[21, 24] discovered the venous valves The name "trephine," he thought, came from its triangular shape Botallus[9] described the foramen ovale, and the presence of foreign bodies in the brain Thomas Gale[28] wrote a treatise on gunshot wounds, the first English book on military surgery

The brain abscess resulting from penetrating head injuries was recognized, but such suppurating complications were left alone Opinion on

the wisdom of removing bone fragments from fractured skulls was divided Dyes were used to detect fissures, as in the Hippocratic era.

Precontemporary Period

The 250 years from the beginning of the seventeenth century to the middle of the nineteenth century can be regarded as the introduction to the modern era. The enormous scientific achievements of the seventeenth century laid a firm foundation for progress in all disciplines. Merely a list of the names gives one a sense of spectacular creativeness Harvey Newton, Bacon, Descartes, Willis, Leeuwenhoek, Malpighi, Galileo Kepler

The medical landmark was the publication of William Harvey's[illegible] work on the circulation of the blood Thomas Willis's[illegible] work on the vascular system of the brain, published in 1664, was illustrated with copperplate engravings, the first time this type of illustration was used. Leeuwenhoek's microscope made possible Malpighi's[illegible] studies in histologic anatomy In 1667 appeared Richard Wiseman's[illegible] text on surgery, including the treatment of gunshot wounds, John Browne's[12] treatise *Complete Discourse of Wounds* appeared a few years later

There was general agreement at this time that suppurating wounds should be drained by opening the skull According to the French surgeon, Pierre François Percy,[illegible] the gunshot wound was to be incised as soon as possible, and he was the first to use the term "debridement to describe this method of wound management. The incision was made to avoid the creation of tension and to prevent strangulation of neighboring tissues by the edema resulting from inflammation along the tract of the missile.

(Wepfer,[illegible] in 1681, recognized chronic subdural hematoma.) In 1699, de La Peyronie[illegible] reported the first case of subdural abscess after head injury (fracture of the left parietal region) He used the trephine and then incised the dura, considerable pus was released and the patient recovered.

Scientific developments continued in full flood in the eighteenth century The list of significant contributors is almost too long to give. Priestley, Volta, Franklin, Celsius, Watt, and Fulton, in medicine, Morgagni, Hunter, Jenner, Linnaeus, von Haller, Whytt, Petit, Heister, Cheselden, Pott, the Monros, and Desault, in the special field of head injuries, Heister, Pott, Bell, Hey, Abernethy, Larrey, and Hilton.

The precepts of the last-mentioned group form the basis for our modern management of head injury, and are therefore important Nevertheless, successful treatment was limited, since wounds commonly became infected whether operation was used or not The importance of the cranial contents began to be recognized, but the main concern was the management of the injuries of scalp and skull and of the extradural collections of blood and cerebrospinal fluid The importance of the dura and the need to avoid its injury were generally recognized, only after knowledge of asepsis gained firm footing, however, did the management of head injury take the long step forward to surgical procedures on the intracranial contents as well as on the skull itself.

A few exceptions may be noted, however Petit[66] performed a successful operation for a brain abscess following a penetrating head injury, and Hill[43] a successful operation for bilateral extradural hematoma

Lorenz Heister[40] discussed contusions and lacerations of the scalp, and skull fractures, which he divided into fissures, contrafissures, and depressions He described the possible extravasations of blood into the cranial cavity after a violent blow, and believed that these could cause the unconsciousness or coma, paralysis, vomiting, and could be associated with bleeding from the mouth, ears, and nose, and inflamed or swollen eyes

For head injuries without serious symptoms he advised the same treatment as for wounds elsewhere Bleeding from the scalp was controlled by a dressing of dry lint held in place by a tight bandage, in some cases, alcohol or other styptics might be used Bags stuffed with medicinal substances and boiled in wine were also recommended

He emphasized the importance of avoiding trephination over the

sagittal suture so as to avoid injuring the longitudinal sinus, and advised against perforating the frontal sinuses. In depressed fractures, free fragments of bone were to be removed, but care was required with deeply embedded fragments. He thought they should be left alone, especially if they could not be removed easily In infants and children with depressed fractures, application of a viscid plaster and exertion of pull on it might raise the depressed area. Trephination was recommended for intracranial extravasation of blood, and for gunshot wounds of the head For subdural hematoma, he advised incision of the dural membrane. He recognized the lucid interval in certain forms of head injury He described extradural, subdural, subarachnoid, and intracerebral hematomas. He stated that a hematoma over the cerebellum was instantly fatal

The English translation of Heister's book probably influenced Percivall Pott,[40] of St. Bartholomew's Hospital, one of the century's great surgeons, whose work on wounds and contusions of the head, skull fractures, and concussion was published in 1760. For a scalp wound in which a good portion of the scalp was torn loose, he advised that it be cleansed of all dirt and foreign matter, after which the loose skin could be restored to its position and held in place by sutures, plasters, and bandages a stitch with a slip knot was used. A sharp blow might so injure the scalp as to cause a hematoma to form, the bordering scalp area might then present a sharp edge, and care was necessary not to mistake this for a depressed fracture. Skull fractures might occur with or without depression, he pointed out, and he listed the usual signs of head injury vomiting giddiness unconsciousness, loss of speech and voluntary motion, bleeding from the ears, nose, and mouth But he was among the first to point out that some of the symptoms were caused by the injury to the brain and the intracranial bleeding, rather than by the skull trauma. A depressed fracture with extensive fissures might occur without any symptoms He noted the possibility of severe mental derangement immediately after the injury, or of a progressive deterioration after a normal interval

(In simple fractures without depression, trephination was performed to obtain immediate relief of symptoms arising from the pressure in the intracranial space) Trephination was also indicated for the discharge of purulent matter formed between the cranium and the dura matter, and for different forms of intracranial hemorrhage Bloodletting was the customary treatment in the conservative management of head injury

Depressed fractures Pott treated by removing those pieces of bone which were completely separated and incapable of reunion and by elevating the remaining fragments with an elevator so that they were properly aligned and did not press on the brain The trephine might be useful for removing normal bone next to the depressed area, if that were necessary A collection of blood in the extradural space was to be removed Bone fragments could pierce the dura, foreign matter could be introduced into the cranial cavity, fragments of bullet could be dispersed throughout the injured area Foreign matter and any free fragments of bone were to be removed if it could be done easily and without force, and the wound dressing, which usually consisted of linen soaked in wine held in place with another dressing, should be applied in such a manner as to encourage drainage

He described what he called "puffy tumor" — actually an extradural abscess — and reported the successful treatment of five cases Probably his treatment succeeded because he resorted to operation before the underlying arachnoid had become involved.

In his discussion of concussion, Pott stated that if unconsciousness followed the injury immediately, it was probably due to a concussion, but when it occurred some time later, extravasation of blood was the most probable cause He described both fluid and organized blood clots in the space between the dura and pia, and undoubtedly also recognized cerebral hygroma If dura pushed into an opening made with the trephine, he advised a crucial incision, although he admitted that while such an incision could release collected blood or fluid a serious situation

might develop Alarming symptoms and death could occur in concussion and commotion, even in the absence of fracture or extravasation as established by postmortem examination

John Hunter[18] was influenced by two great surgeons — William Cheselden of St. Thomas's Hospital and Percivall Pott of St. Bartholomew's. Hunter's pupils, in turn, included Jenner, Cooper, Abernethy, Cline, Cleft, Parkinson, and Physick. All of them made significant contributions to the development of surgery and in the early years of the nineteenth century the leadership in this branch of medicine passed from the French to the English.

The list of those who made significant contributions and advanced the borders of knowledge in the sciences, including medicine, grows long Not only the treatment of head injuries but the mechanism of their production, was the subject of study and discussion in the eighteenth and the first half of the nineteenth century An account of some of this work may be found in Dulles.[1] Leading men interested in head injury toward the end of the precontemporary period included the Bells, Cooper, and Abernethy in Great Britain, Larrey, Dupuytren, and Desault in France, Dieffenbach Stromeyer, and von Bergmann in Germany Pirogoff in Russia, Physick, Post, and McDowell in America.

John Bell,[6] in his *The Nature and Cure of Wounds* argued against the practice of cicatrizing wounds and of allowing wounds to fill with proud flesh to the surface. For head wounds without signs of brain trauma he advised pulling the cut edges of the scalp together and suturing, even if there were no bone underlying the skin He cited Berengarius (Berengario da Carpi)[7, 71] as having successfully used this method in the sixteenth century In contrast to Pott, Charles Bell,[8] and others of his time, he was opposed to the use of the trephine except for depressed fractures. Concussion he described as an affection of the whole nervous system, and listed the symptoms dilated pupils, slow pulse, slow stertorous breathing, cold and possibly convulsed extrem-

ities, and unconsciousness. He thought that concussion could be fatal. He correctly differentiated between concussion and compression due to hemorrhage or pressure by depressed fracture

John Abernethy,[1] a pupil of Hunter and his immediate successor, recorded his observations on head injuries in 1811. He described extradural and subdural hematomas, discussed hernia cerebri, and pointed out that trephination was successful only in the treatment of extradural collections of blood or fluid He also discussed concussion and cerebral inflammation

Sir Astley Cooper,[17] in his *Lectures on the Principles and Practice of Surgery,* which appeared in 1824, discussed the mechanism of concussion and suggested that it might be due to a change of function rather than to a disorganization of the brain In slight concussion it was possible that only the cerebral circulation was disturbed, whereas in severe concussion the brain might be torn Bloodletting was part of his treatment of concussion, but not trephination He also emphasized the difference between concussion and compression Bloodletting and purgatives were the treatment of choice for skull fractures accompanied by signs of brain injury.

For depressed fractures he advised conservative treatment if the scalp was not lacerated For open fractures, with their danger of resulting brain inflammation, he thought the depression should be elevated His indications for the use of the trephine were sound. (*1*) when there is extravasation of blood between the dura mater and the skull, (2) in skull fractures with symptoms of compression, (*3*) in simple fractures with depression and continued symptoms of compression; (*4*) in compound fractures with depression, even in the absence of compression symptoms, and (*5*) when matter had formed. In his discussion of brain trauma, he pointed out that hemiplegia is often on the side opposite to the injury

Cooper made an interesting experiment, comparable to that reported by Cushing, and later by Browder and Myers, which he described as follows:

In order to ascertain the symptoms arising from depression, I tried the following experiment. I applied the trephine to the cranium of a large dog and took out a portion of bone. I then with the handle of a knife separated the dura mater from the bone for I found that I could make no impression on the brain until I had done so, and then pressed upon it with my finger. At first the animal did not seem to feel it but upon pressing more deeply it produced pain and irritation, and he endeavoured to avoid it. Upon still increasing the pressure, he became comatose and sunk on the table. I kept him in this state for five or six minutes when, upon removing my finger, he got up, turned around two or three times from giddiness, and walked away apparently little worse for the operation. A gentleman who felt the animal's pulse during the continuance of the experiment, stated that it became slower as the pressure was increased. In depression of the skull in man the pulse is the same—slow and labouring and the breathing is often stertorous.

Dominique Larrey[41] was a military surgeon and his experience in the Napoleonic Wars was vast. In his opinion, the treatment for a clean, simple division of the skin caused by firearms was closure with a piece of fine perforated linen dipped in warm, sugared or honeyed wine and spreading a balsamic substance over the surface in order to keep the edges together. Adhesive strips were applied to extensive and ragged cuts, and then the dressing placed over them. A few stitches might be necessary in some cases, but an opening should be left to facilitate drainage of fluids collecting under the skin. The trephine was indispensable in treating skull fractures in which bone fragments might be displaced and driven into the brain or when foreign substances were present in the brain. If foreign bodies could not be removed without further damage to the brain, they were to be left in place. Trephination might also be necessary in case of hemorrhage. Operation should be done as soon as possible, since after 24 hours or more inflammation usually developed in head wounds, and the trephine was then useless. He explored wounds with a probe, but only when he was fairly certain that the dura was intact. Hernia cerebri, he thought, was due to a deep-seated inflammation within the parenchymatous substance, and the condition was most difficult or impossible to cure.

Dupuytren,[22] in 1841, successfully treated a subdural abscess in a patient who had been wounded by a knife two years previously and was comatose and hemiplegic on the side opposite the injury After trephination, which yielded no pus, Dupuytren incised the dura and the brain substance, which released a large quantity of pus Several reports of such abscesses were made during the century.[35, 37, 47 67 77]

Like Cooper and Larrey, Guthrie[37] thought the trephine should be employed to remove indriven bone fragments and effused fluid He advised examination of cranial wounds with a blunt, flat probe to determine the extent of damage In the absence of a scalp wound, one might have to depend on the appearance of paralysis, most commonly on the contralateral side from that of the brain injury, before proceeding with exposure by the trephine and surgical treatment. He reported two cases of subdural abscess after head injury

For a time after the Napoleonic Wars, penetrating wounds of the head were treated more conservatively Longmore[52] thought trephination useless and even dangerous except in cases with indriven bone fragments or for evacuation of an abscess This was also the attitude of Stromeyer,[72] a German military surgeon, and of Macleod,[53] whose experience in the Crimean War led him to state

. The teaching of all was to lead us to wait; to purge the patient thoroughly; to remove only such pieces of bone as could be got at with the forceps, and which were quite detached and loose, to bleed, if need be, locally and even generally, to use cold applications when there was a fear of inflammation, to enjoin perfect rest not only to the body generally, but, if possible, to give repose to the special senses also, by isolating the patient, and thus removing the stimuli to their exercise, to enforce the lowest diet, and to continue all this treatment for a long period, even after all danger seemed past, and, finally, to treat any incidental complications on general principles

This may be regarded as the view of most surgeons of the midnineteenth century

Although Lister's principles of antisepsis became known in 1867, the

treatment of head wounds in the Franco-Prussian War (1870) and the Russo-Turkish War (1877) continued much as before.

The knowledge and methods of treatment of head injuries, as summarized by Hewett[41] in 1861, reveals a surprising amount which is still acceptable by modern standards. For example, scalp wounds were carefully cleansed, and the flap, however extensive, was replaced and held in place by plasters, compresses, or bandages, and a few sutures, if necessary. Any pus under the scalp was drained. Loose fragments of bone were removed. The trephine was rarely used. The management of a depressed fracture depended on the presence or absence of an overlying laceration. Operative repair of open depressions was done as early as possible, except in children and infants, in whom treatment could often be conservative. A closed fracture, *i.e.* a simple depression, was treated conservatively.

Discharge of blood and cerebrospinal fluid from the ear after head injury, Hewett noted, had been described as early as 1727; a watery discharge from the nose was noted in 1840 in a report from the Hôtel Dieu and the source of the rhinorrhea was definitely identified by autopsy.[20]

Management of intracranial hemorrhage was limited mainly to collections between the skull and the dura. An occasional subdural hematoma was also drained. Infection after opening the dura was often fatal, and the importance of leaving the dura intact in instances where it was not torn was therefore emphasized.

There was evident interest in the mechanism of head injury in the eighteenth and nineteenth centuries. As early as 1769, the French Royal Academy of Surgery offered a prize award for an essay on "the theory of counter stroke in lesions of the head and the practical conclusions that may be drawn from it."[26] In England, too, men like Charles Bell[4, 5] and George Guthrie[37] began to experiment and speculate on the subject. On the basis of his experimental observations, Bell concluded that a blow to the skull shortens the diameter parallel to the direction of the force and lengthens the one perpendicular to it. Aran[3] experimented

with skulls from cadavers and dry skulls and evolved the "radiation theory" of skull fracture According to this theory, a fracture of the vault radiates to the base, taking the shortest route or the curve of shortest radius, even crossing sutures Hilton[44] discussed the role played by the cerebrospinal fluid cisterns and by the configuration of the base, particularly of the petrous bone, in dissipating the force of a blow to the skull, in his opinion, these aided in damping the conduction of "vibrations" to the brain. Von Bruns[14] described the elasticity of the skull

Hewett[41] divided skull fractures into vault and base fractures, and the latter into direct and indirect Like 'Aran, he thought indirect basilar fractures uncommon, indirect fractures might be caused by a thrusting up of the spinal column or by the thrust of the condyloid processes or the lower jaw against the base of the skull.

Félizet[26] reported that indirect fractures resulted from a disruption by splitting, and that the course of a fracture is determined by the bone reinforcements, particularly the buttresses. midline frontal and occipital, and lateral parietosphenoid and parietopetrous He denied the validity of the vibration theory, believing the important mechanism to be a violent flattening of a portion of the vault, with separation of the resisting supporting parts, *i.e.*, the buttresses. Fractures caused by impact perpendicular to the cranial surface would radiate to the base

P von Bruns[13] suggested that basilar fractures were the result of the special fragility of this portion of the skull. Messerer[56] demonstrated that compression of the skull in one axis enlarges the circumference at right angles to it He noted that indirect or contrecoup fractures resulted from separation in the meridional plane, due to the depression at the point of impact His work, and the earlier observations of V von Bruns are particularly important in explaining the mechanism of head injuries by static loading, as, for example, in birth injuries

Von Wahl[76] suggested that fractures may arise as a result of: (*1*) a crushing injury, in which the fracture line runs at right angles to the axis of the force, or (2) bursting, in which the fracture lines are parallel

to the axis of the force. Because of the skull's elasticity, gradual compression would produce results different from those of sudden or acute compression.

Littre, in 1705,[81a] described a fatal case of "concussion" in a prisoner who ran head first into a wall to escape punishment Examination of the patient's head showed the brain to be shrunken, without other lesions. During the following 150 years, concussion or immediate post traumatic unconsciousness was explained on the basis of intracranial commotion following an impact. "Functional disturbance" involving the entire brain was the explanation for concussion by Cooper[17] Gama[40] studied mass movements by the use of threads in a jelly to simulate conditions at impact involving the head The more exact study of the pathophysiology of head injury was to make its beginning in the latter part of the nineteenth century

Contemporary Period

While this period stretches from the second half of the nineteenth century to the present day, only the beginnings of it will be briefly sketched here. The remainder is essentially a part of all the rest of this book, and will be found in appropriate places throughout our discussion of the mechanisms, the treatment, and the complications of head injuries.

The great names that usher in our own times are, among others, those of Louis Pasteur, Joseph Lister Claude Bernard, Rudolf Virchow, and Robert Koch. Their contributions to our knowledge of bacteriology, antisepsis, cerebral physiology, and cellular pathology made possible the rapid advances in all fields of medicine and surgery In cranial surgery, Ernst von Bergmann,[8] Victor Horsley,[61] Fedor Krause,[18] and Samuel Gross[54] all developed important technics and methods.

The two world wars, and the greatly improved technics of experimental study of our own century have brought with them a great

expansion in the knowledge of the why's and wherefore's of head injuries, and in what to do about them

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Chapter II

ANATOMIC CONSIDERATIONS

Certain anatomic features of the head and craniospinal junction are important in connection with the mechanisms and the causation of the signs and symptoms of head injury. Most of the pertinent information is outlined in this chapter, as it applies to the problems of head injury, some additional references to anatomy will be found in chapters discussing special problems, *e.g.*, head injury in infants and children. In so far as it seemed necessary for ease of understanding, the anatomy and physiology of the brain are considered in the several chapters on the clinical aspects of head injury

Scalp

The scalp is a 5 mm thick structure composed of 5 layers: skin, subcutaneous tissue, galea aponeurotica, subaponeurotic space, and the periosteum. The loose subaponeurotic space and the frontalis and occipitalis muscles are responsible for the mobility of the scalp The three outer layers are closely adherent and move as a unit. The galea aponeurotica is attached to the frontalis muscle anteriorly and the occi-

pitalis muscle posteriorly, these muscles serve the volitional scalp movements.

The scalp has an abundant blood supply The arteries are the supraorbital, frontal, temporal, posterior auricular, and occipital, with extensive anastomoses between the vessels of the two sides. Veins accompany the arteries and empty into the veins of the face, and the external jugular and anterior jugular groups. Some of the veins in the occipital area and about the ears empty into the deep cervical and the vertebral venous channels.

Skull and Intracranial Structures

Skull

The skull is a spheroid structure, made up of the vault and the base, enclosing brain, blood, and cerebrospinal fluid It is a closed cavity solidly supported by bone in all directions except at the foramen magnum. This structural characteristic is an extremely important factor in injury to the brain stem centers by blunt head injuries (page 96)

In the adult, the thickness of the skull averages 2 to 6 mm. It is much thicker in the vicinity of the buttresses, and much thinner around the temporal fossa sometimes almost paper thin. In cross section, the vault is seen to consist of a solid outer layer, a cancellous middle portion (diploe), and a solid inner layer As a rule, the outer layer is twice as thick as the inner but occasionally the inner layer may be thicker than the outer one as a result of diploic ramifications.

The diploe contains the diploic veins the frontal the anterior temporal, the posterior temporal and the occipital. These veins communicate with the large venous sinuses intracranially and the extracranial veins externally

The skull is also traversed by emissary veins which are direct communications between the intracranial venous sinuses and the extracranial venous channels (*1*) the parietal and frontal emissary veins with the sagittal sinus internally and the scalp veins externally, (2)

the mastoid emissary vein with the transverse sinus internally and the scalp veins of the neck and mastoid area externally. In some skulls, there may be other emissary veins as well

The base of the skull is a membranous structure with a cancellous or diploic layer only in certain areas The three main divisions of the base are the anterior, the middle, and the posterior fossae The anterior fossa is formed by the horizontal or orbital portion of the frontal bone laterally, the lesser wing of the sphenoid and the body of the sphenoid posteriorly, and the cribriform plate of the ethmoid in the midline. Projecting upward from the midline of the cribriform plate in its forward portion is the crista galli, to which is attached the falx cerebri. The middle fossa is formed of the greater wing of the sphenoid anteriorly, the body of the sphenoid (containing the pituitary fossa) in the middle, and the squama and the anterior aspect of the petrous portion of the temporal bone laterally and posteriorly The anterior and middle fossae are separated by the sphenoid ridge, with its sharp, knife-like edge fitting against the junction of the frontal and temporal poles of the hemispheres The posterior fossa is formed by the posterior aspect of the petrous portion of the temporal bone anteriorly and the occipital bone posteriorly The foramen magnum is in the center of the posterior fossa, with the basilar process of the occipital bone and the posterior part of the body of the sphenoid forming the base of the skull in front of the foramen magnum.

Buttresses and Shape of Skull

Félizet,[7] in 1873, described the cranial buttresses as 6 in number: single midfrontal and midoccipital and paired parietosphenoid and parietopetrous buttresses The bone is thicker in the region of the buttresses; a fracture line progressing along the side of the reinforced bone tends to extend into thinner bone, whereas a fracture line at right angles to a buttress bisects it without difficulty

The stress characteristics of the skull are affected by the thickness and strength of the bones, as well as by its shape Variations in the

shape of the head, therefore, can cause differences in the fracture pattern The stress characteristics of an abnormally long and narrow skull (scaphocephaly) such as results from early closure of the sagittal suture, differ from that of the rounded skull (turricephaly), or from that of an asymmetric skull (plagiocephaly)

Dura Mater and Piarachnoid

The dura mater serves a double function it is the periosteum of the inner aspect of the skull and a protective membrane for the brain. It consists of white fibrous tissue and yellow elastic connective tissue and is separated into two layers — the endosteal, which is the intracranial periosteum, and the meningeal which is in juxtaposition to the cranial contents. The two layers are closely adherent except in certain places where they separate to form the venous sinuses. The endosteal layer adheres closely to the inner surface of the bones. The dura mater is prolonged to the outer surface of the skull through the foramens at the base, and thus becomes continuous with the pericranium At the foramen magnum the dura mater adheres closely to the bone and is continuous with the dura mater of the spine. Four processes extend inward into the cranial cavity, formed by reduplication of the meningeal layer the falx cerebri the tentorium cerebelli, the falx cerebelli, and the diaphragma sellae.

The arteries of the dura mater are (*1*) in the anterior fossa, branches from the anterior and posterior ethmoidal arteries, from the internal carotid artery and from the middle meningeal artery, (*2*) in the middle fossa, branches from the internal maxillary and from the ascending pharyngeal arteries some twigs from the internal carotid artery and a recurrent branch from the lacrimal artery (*3*) in the posterior fossa, branches from the occipital and vertebral arteries, an occasional branch from the ascending pharyngeal artery, and a branch from the middle meningeal

The veins of the dura mater anastomose with the diploic veins and end in the venous sinuses. Some of the veins do not open directly into

the sinuses but indirectly through a series of venous lacunae, along each side of the sagittal sinus, particularly of its middle portion, and near the transverse and straight sinuses After the age of 6, arachnoid granulations extend into the venous lacunae, particularly those along the superior sagittal sinus.

The falx cerebri, a sickle-shaped fold of the meningeal layer, is located in the midline and in a sagittal plane between the cerebral hemispheres It is attached to the crista galli anteriorly and blends with the upper surface of the tentorium cerebelli posteriorly The sagittal sinus is located at the junction of the falx cerebri with the internal surface of the skull The inferior sagittal sinus lies along the free margin of the lower border of the falx Its attachment to the tentorium helps to form the straight sinus.

The tentorium cerebelli, a transversely placed structure, separates the posterior portions of the cerebrum from the cerebellar lobes Its concave, free anterior border surrounds an opening, the incisura tentorii, for the passage of the brain stem. Laterally and posteriorly, the tentorium is attached to the skull along the sinus impression on the interior surface of the occipital bone, where it encloses the lateral sinuses. Anteriorly, it is attached to the posterior clinoid process and the superior border of the petrous part of the temporal bone on both sides, enclosing the superior petrosal sinuses

The falx cerebelli is a small evagination of dura mater between the two cerebellar lobes and contains the occipital sinus

The diaphragma sellae is a small, circular fold which covers the sella turcica; a small opening posteriorly permits the passage of the infundibulum from the hypothalamus to the gland

The arachnoid, a thin membrane made up of white fibrous and yellow elastic tissue, lies between the pia mater and the dura mater It is almost avascular, and its outer and inner surfaces are covered with a low cuboidal mesothelial lining It covers the brain loosely but does not dip into the sulci between the convolutions. Over the convexity

of the hemispheres the arachnoid is thin and transparent at the base it is thicker and opaque.

Within the subarachnoid cavity, the space between the arachnoid and the pia mater, a tissue consisting of trabeculae of connective tissue and intercommunicating channels holds the subarachnoid fluid The cavity is much more evident in the sulci than over the convolutions, where the arachnoid and the pia mater are close together At certain parts of the base of the brain, wide intervals, the subarachnoid cisterns, separate the arachnoid from the pia mater

The pia mater is a vascular membrane of connective tissue covering the entire surface of the brain and dipping down between the cerebral convolutions. Over the cerebellum the membrane is much more delicate and is less intimately related to the cerebellar surface than it is to the cerebral hemispheres.

Intracranial Calcifications

A number of calcifications are normally present. The calcified pineal gland is important, being found in 50 to 60 per cent of adults and varying in diameter from 1 to 5 mm., it may be displaced by a space occupying lesion The habenular calcification is a C-shaped shadow directly in front of the pineal, with the open end of the *C* directed posteriorly It, too, may be shifted, but its position about 6 mm in front of the pineal and in about the same position in the center of the head should be kept in mind Calcific deposits in the falx cerebri and on the dura are often seen, in some instances, displacement of such a deposit may indicate the side on which a mass lesion is present. Pacchionian bodies near the midline of the vertex appear as small shadows of 5 to 10 mm. diameter in relation to the pacchionian granu lations. Calcifications in the diaphragma sellae, the tentorium cerebelli, and the petroclinoid ligament may also be present.

Paranasal Sinuses

The paranasal sinuses include the frontal, ethmoid, sphenoid, and the maxillary The frontal sinuses are practically absent at birth, while the ethmoid and maxillary sinuses are of fairly good size although not aerated They become aerated during the first few weeks of life, although the maxillary sinus is not fully developed until the age of 7 The sphenoid sinus begins to form about the third year as an evagination of the posterior superior portion of the nasal cavity into the body of the sphenoid bone

The *frontal* sinuses are usually 2 in number, separated from each other by a septum, occasionally, there are two or more sinuses on each side, connecting with the nose by separate ostiums or intercommunicating with each other. The size of the frontal sinuses varies from large enough to involve the entire frontal region from temple to temple and extend into the vault for 4 to 6 cm to very small, or, a sinus may be lacking on one side The sinuses communicate with the nose through the frontonasal duct, or through a separate ostium emptying into the nose, or through the anterior ethmoidal cells into the nose The inner wall of the frontal sinus forms part of the base of the skull and the floor of the anterior fossa

The *ethmoid* sinuses or cells range in number from 3 to 18, but usually are 10 or more on each side of the midline between the medial orbital wall and the lateral aspect of the upper nasal border They are divided into an anterior and posterior group, the anterior cells empty into the frontal recess, the ethmoidal infundibulum, or directly into the middle meatus; the posterior cells usually open into the superior meatus The cells extend from the region posterior to the frontal sinuses to the anterior border of the sphenoid sinuses

The *sphenoid* sinuses, usually 2 in number, one on each side, vary in size and shape They empty into the posterior nares above and behind the superior nasal concha. and occasionally communicate with each other. In some, the sinus may extend into the lesser wing of the

sphenoid and completely encircle the optic canal, or into the greater wing of the sphenoid, or into the pterygoid process and posteriorly into the basilar process of the occipital bone.

The *maxillary* sinus or antrum lies within the body of the maxilla the floor of the sinus is the roof of the mouth its anterior wall is part of the face, posteriorly it is bounded by the infratemporal area, and its roof is the floor of the orbit. The antrum usually measures about 3×2×3 cm., but the cavity may be smaller or larger The sinus empties through an opening into the ethmoidal infundibulum immediately under the middle turbinate

Ethmoid Bone

This bone consists of four parts a perpendicular plate, a horizontal plate or cribriform plate, and two lateral masses or labyrinths. The perpendicular plate forms part of the nasal septum. The labyrinths consist of the ethmoidal cells the lateral surfaces are formed of the laminae papyraceae, which in turn also form the medial walls of the orbits. The horizontal or cribriform plate forms part of the base of the anterior fossa in the midline. The crista galli rises from the middle of the cribriform plate, and serves for the attachment of the anterior end of the falx cerebri, the deep grooves of the plate on either side of the crista galli contain the olfactory bulb and foramens for the *passage of the olfactory nerves.* The anterior and posterior ethmoidal foramens serve for the passage of blood vessels and other nerves.

Orbit and Its Contents

The bony structure of the orbit consists of (*1*) the roof formed by the orbital plate of the frontal bone anteriorly and the lesser wing of the sphenoid posteriorly (2) the floor, formed mainly by the orbital surface of the maxillary bone, by the orbital process of the zygomatic bone anteriorly and laterally and by the orbital process of the palatine bone medially and posteriorly, (*3*) the medial wall, formed by the frontal process of the maxillary bone, the lacrimal bone, the lamina

papyracea (a very thin wall separating the ethmoidal air cells from the orbit), the ethmoid, and a small portion of the sphenoid body, (*4*) the lateral wall, formed by the orbital surface of the greater wing of the sphenoid and the orbital process of the zygomatic bone. More posteriorly, in the vicinity of the optic foramen, the lesser wing of the sphenoid contributes to the lateral aspect of the optic opening.

The optic foramen, or apex, which affords passage for the optic nerve and the ophthalmic artery, is a 4 to 10 mm. long canal between the body and the lesser wing of the sphenoid. On its medial aspect are the sphenoid sinuses, or, if the latter are not well developed, the posterior ethmoid sinuses. The sphenoid sinuses may extend into the greater wings of the sphenoid, thus completely surrounding the optic canal. The same relationship to the canal may occur if the ethmoid sinuses are overdeveloped.

The superior oblique muscle arises above the margin of the optic foramen and ends in a cartilaginous structure attached to the trochlear fovea. The inferior oblique muscle arises from the floor of the orbit just posterior to the orbital rim near the opening for the nasolacrimal canal. The common tendon around the optic foramen gives origin to the rectus muscles: the superior, inferior, lateral, and medial recti. The supraorbital fissure lateral to the optic foramen affords passage of the third, sixth, and fourth cranial nerves, the ophthalmic division of the fifth cranial nerve, and sympathetic nerve fibers and the ophthalmic veins.

The orbital fasciae include the periosteal lining, also called the periorbita, which helps to form the orbital septum anteriorly and the tendon of Zinn posteriorly. This lining is continuous with the dural lining intracranially at the various foramens. Tenon's capsule, a layer of fibrous tissue surrounding the orbital fat of the eyeball, is immediately internal to the periorbita. Extensions of the capsule sheath the muscles surrounding the eyeball.

The optic nerve, together with the piarachnoid and the dura, traverses the optic canal. A subarachnoid space usually accompanies

the optic nerve and extends to the lamina cribrosa, at which point the arachnoid and dura become attached to the scleral lining of the eyeball The orbital portion of the optic nerve, 20 to 30 mm long follows a somewhat tortuous course, to allow for the movements of the eyeball. In its course through the canal, the ophthalmic artery is inferior and lateral to the optic nerve. The length of the nerve between the canal and the optic chiasm is about 10 mm. Just before it enters the eyeball the nerve is pierced by the retinal artery, and nerve and artery pass into the interior of the eyeball. The anatomic distribution of the optic radiation is described by Spalding[7] After leaving the lateral geniculate body, optic fibers tend to go forward and then bend posteriorly forming Meyer's loop in the depth of the posterior temporal lobe, they then extend toward the calcarine cortex on the lateral aspect of the ventricular body vertically and in the form of a horseshoe, with the open end toward the ventricle along the posterior horn.

The eyeball is enclosed within Tenon's capsule, surrounded by muscles, nerves, and periorbital fat. The eyeball is made up of 3 layers the sclera, the choroid, and the retina The choroid forms the iris, with its centrally located pupil, and the sclera forms the transparent cornea in front of it. The anterior chamber of the eye contains the aqueous humor, the posterior chamber the viscid vitreous. Although the eye ball is quite tense, there is a certain amount of give to it. The nerves that pass through or end in the orbit include the ophthalmic division of the trigeminal and the oculomotor, trochlear, and abducens nerves, as well as sympathetic and parasympathetic fibers.

Temporal Bone

In the adult, the temporal bone consists of the squama the petrous portion which contains the hearing and labyrinthine apparatus, the mastoid and the tympanic portions, and the styloid process. The temporal bone articulates with the parietal bone and the sphenoid anteriorly and superiorly the occipital bone posteriorly, and with the mandible and zygomatic bone.

The squama, the anterior and upper portion of the temporal bone, is thin, translucent, and with a smooth outer surface; it serves as attachment for the temporalis muscle The zygomatic process, which houses the mandibular fossa, projects from the lower part of the squama, at the junction with the petrous portion Immediately posterior to the mandibular fossa, on the external aspect of the squama, is the external auditory meatus, made up of a lateral cartilaginous part and a medial bony portion Posterior to the meatus is the mastoid process, a conical projection of the mastoid portion of the temporal bone

The mastoid part of the temporal bone is its posterior part On its outer surface it serves as attachment for the occipital and posterior auricular muscles and more inferiorly for the sternocleidomastoid, splenius capitis, longissimus capitis, and digastric muscles

The petrous portion, or pyramid, is at the base of the skull, between the sphenoid and the occipital bone Its anterior surface forms part of the middle fossa, its posterior surface the front part of the posterior fossa The superior border has a groove for the superior petrosal sinus Along the anterior surface are the hiatus of the facial canal, the impression of the carotid canal, and the tegmen tympani, the thin roof of the tympanic cavity. Posteriorly, there is a sulcus for the sigmoid sinus; in the middle third is the internal acoustic meatus, and in its lateral third, the structures of the inner ear.

Tympanic Cavity (Middle Ear)

This complicated structure consists of a lateral or membranous wall, a medial or labyrinthine wall, a posterior or mastoid wall, an anterior or carotid wall, the tegmental wall or roof, and the jugular wall or floor It contains three ossicles — the malleus, the incus, and the stapes — which are important in the conduction of sound waves when the tympanic membrane is intact. The malleus is attached to the lateral wall, which is largely formed by the tympanic membrane; it articulates with the stapes, which is attached to the fenestra ovalis, an open-

ing in the medial wall. This oval opening communicates with the vestibule. The floor, a thin layer of bone, covers the jugular vein and bulb, the posterior wall communicates with the mastoid cells through the antrum the anterior wall separates the middle ear from the carotid canal and is in communication with the eustachian tube the medial wall, in addition to the fenestra ovalis, has a round opening, the fenestra cochlea or rotunda and a promontory between the two openings.

Acoustic Nerve (Cranial VIII)

This nerve is composed of a cochlear and a vestibular portion. The cochlear nerve transmits sounds, as nerve impulses, from the organ of Corti to the brain. The vestibular, or balancing nerve, carries impulses from the vestibular saccule and utricle and the semicircular canals thus serving to control the orientation of the body at rest or in motion The cochlear duct, a spiral structure communicating with the vestibular saccule and protected by the cochlea, mediates the reception of sound The duct is filled with endolymph and contains the organ of Corti, the receptor organ of hearing

Both the auditory and balancing mechanisms are located in the inner ear or labyrinth It consists of two parts the osseous, which is a series of cavities within the petrous portion of the temporal bone, and the membranous a number of communicating sacs and ducts within the cavity The membranous structures are filled with endolymph, a similar fluid the perilymph lies between the membranous structures and the bony cavities enclosing them. The saccule, the utricle, and the semicircular canals make up the membranous labyrinth. All three structures contain sensory hair cells covered by a gelatinous material containing particles of calcium carbonate, the otoliths, the movements of which stimulate the sensory cells.

The vestibular and cochlear nerves, united in a common body the acoustic nerve, pass through the internal auditory meatus, behind and slightly inferior to the facial nerve, enter the brain stem near the lower lateral border of the pons, and then run separate courses. The primary

cell station of the vestibular nerve is the vestibular ganglion, in the internal auditory meatus The centrally directed fibers end in the four vestibular nuclei: the superior, lateral, medial, and spinal Fibers from the vestibular nuclei join the medial longitudinal or posterior longitudinal fasciculus, and also form the vestibulospinal tract The possibility of a vestibular center in the cortex in the temporal lobe area has been suggested

Fibers of the cochlear nerve, whose primary cell station is the spiral ganglion, on entering the medulla oblongata, connect with the dorsal and ventral cochlear nuclei Other fibers course upward and connect with the inferior colliculus, the medial geniculate body, and finally with the cortical auditory centers in the transverse temporal gyri of the superior temporal convolution Since the fibers arising from the dorsal and ventral cochlear nuclei ascend on both sides of the midline, destruction of the cortical auditory center on one or the other side does not result in deafness

Facial Nerve (Cranial VII)

The seventh cranial nerve (facial nerve or intermediofacial nerve) is composed of a main motor portion and a smaller sensory and visceromotor root, the nervus intermedius Both portions emerge on the lateral aspect of the brain stem and enter the internal auditory meatus along with the acoustic nerve, the nervus intermedius lying between the facial and acoustic nerves In the internal auditory meatus, the facial separates from the acoustic and enters the facial canal, where it lies adjacent to the tympanic cavity At the geniculate ganglion, which contains the cells of origin of the nervus intermedius, the nerve turns from its lateral direction, bending caudally until it reaches the stylomastoid foramen After its exit from this foramen, the nerve enters the parotid gland and then divides into several branches which supply the facial muscles It also sends fibers to the stapedius and the tensor tympani muscles in the middle ear, and to the stylohyoid and the

platysma muscles in the neck. The sensory fibers of the facial nerve mediate the sense of taste in the anterior two-thirds of the tongue via the *chorda tympani nerve*. The visceromotor fibers of the facial nerve help to control lacrimation through the greater superficial petrosal nerve, which branches off at the geniculate ganglion.

Structures and Relationships at Incisura

The incisura is an opening about 40 mm. wide in the tentorium. It extends for about 55 mm. from the clinoid processes on both sides to *the junction of the falx cerebri and the tentorium in the midline*, the point at which the straight sinus begins, with the great vein of Galen emptying into it. A rather large vein nearby, the basal vein of Rosenthal, empties into the great vein of Galen The former usually parallels the course of the posterior cerebral artery around the cerebral peduncle The two posterior cerebral arteries give off several twigs to the brain stem which extend into it at the interpeduncular space. The posterior communicating arteries extend forward from the posterior cerebral artery to the carotid arteries on both sides The third cranial nerves (oculomotor) pass forward to the lateral aspect of the pituitary fossa through the cavernous sinuses, and the fourth cranial nerves (trochlear) also extend in the same direction, more laterally and caudally The anterior peduncles of the midbrain are in a lateral and anterior position and are only 3 to 5 mm. from the incisural edge on both sides The medial portion of the uncus is normally apposed to the incisural opening

Vascular Structures

Arterial blood is supplied by the external and internal carotid arteries and the vertebral arteries, and their branches. The venous drainage is accomplished by the cerebral veins and the dural sinuses, with the diploic and emissary veins functioning as communications between the intracranial and extracranial venous channels.

Middle Meningeal Artery and Veins[5, 6]

This artery is a branch of the external carotid through its internal maxillary branch It is accompanied by 2 or 3 veins in its intracranial course and supplies the greater part of the dura in the middle two-thirds of the cranium. It enters the cranium through the foramen spinosum in about 57 per cent of cases, and either divides into an anterior and posterior branch immediately, or runs along the greater wing of the sphenoid bone for 1 to 5 cm before dividing into the two branches The anterior branch is usually larger, and appears to be a continuation of the main trunk In some, the anterior branch may be given off from the ophthalmic artery, and it may then enter the cranial cavity through the supraorbital fissure; in such cases, the foramen spinosum may be absent, or it may be a passageway for a smaller artery, the equivalent of the posterior branch. In 60 of 100 cases studied, the anterior division extended through the greater wing of the sphenoid for 1 to 3 cm in a bony canal[6]

In the adult, the meningeal artery is partly encased in a groove on the inner surface of the skull; these grooves are absent or are extremely shallow in infants and children.[7]

Internal Carotid Artery and Vertebral-Basilar Complex

The internal carotid arises from the carotid bifurcation at the upper border of the thyroid cartilage and courses upward in front of the transverse processes of the upper 3 cervical vertebrae After it enters the carotid canal, the artery at first courses along the anterior aspect of the tympanic cavity, then medially and forward until it pierces the dural lining on the side of the sphenoid body, where it forms a *U*-shaped structure known as the siphon. The ophthalmic branch is given off as the artery enters the dural sac, and several other branches after it pierces the dura mater: the anterior and middle cerebral arteries, the posterior communicating artery, and the anterior choroidal artery

The stem of the internal carotid may be compressed by increased intracranial pressure and cerebral edema, edema of the temporal lobe with resultant medial compression, particularly, distorts this vessel

Anterior Cerebral Artery

It courses forward from its origin to the longitudinal fissure, passing over the optic foramen and the anterior perforated area. At the fissure it connects via the anterior communicating artery with the anterior cerebral from the opposite side. A branch is given off just before this point — Heubner's artery also known as the medial striate artery or the recurrent branch — which supplies the anterior portion of the internal capsule and the caudate nucleus. Other anterior striate branches supply the hypothalamus The two anterior cerebral arteries run side by side through the fissure, around the genu of the corpus callosum, and then along the upper surface of this structure to its posterior aspect. Inferior (frontopolar), middle (callosomarginal), and posterior (pericallosal) branches are given off The inferior branches supply the orbital surface of the frontal lobe, the middle branches, the corpus callosum and the paracentral lobule and the posterior branches, the precuneus and the neighboring lateral surface of the hemisphere.

The anterior cerebral artery may be distorted as a result of increased intracranial pressure, particularly in its relation to the falx cerebri and the corpus callosum

Middle Cerebral Artery

This artery is a rather direct continuation of the internal carotid It first courses in the sylvian fissure, then on the surface of the insula where it divides into several branches (*1*) the lateral striate, supplying the basal nuclei and the internal capsule, (*2*) the inferior lateral frontal (*3*) the ascending frontal, (*4*) the ascending parietal (*5*) the parietotemporal and (*6*) the temporal

Posterior Cerebral Artery

This artery is a branch of the basilar artery, but in a small percentage of cases it may be a branch of the internal carotid. The artery circles around the cerebral peduncle and reaches the inferior surface of the occipital lobe. Small branches supply the posterior aspect of the thalamus and hypothalamus Anterior and posterior temporal branches are distributed to the uncus and the more posterior portions of the inferior temporal gyrus A calcarine branch supplies the cuneus; a parieto-occipital branch, the cuneus and the precuneus.

Involvement of the posterior cerebral artery by deformation of the brain stem due to increased intracranial pressure, particularly associated with temporal lobe or uncal herniation, may result in extensive infarction of the occipital portion of the cerebral hemisphere, on one or both sides.

Among the branches of the vertebral and basilar complex, the superior cerebellar arteries may be compressed and distorted by uncal herniation, the pontine branches of the basilar may compress the abducens nerve, the anterior-inferior cerebellar and the posterior-inferior cerebellar arteries may be involved in herniations about the foramen magnum

Arterial Supply of Pituitary Gland

According to Dawson,[2] the pituitary gland and its stalk are supplied by the superior hypophysial arteries which arise from the intracranial portion of the internal carotid artery, by the infundibular branches arising from the posterior communicating arteries, and by connecting vessels from an anastomosis on the anterior aspect of the optic chiasm The anterior pituitary is supplied by two sets of vessels from this anastomosis, one extending along the surface of the stalk and the other forming the portal system within the stalk. The posterior pituitary lobe is supplied from the internal carotid arteries in the cavernous sinuses

Veins

The *cerebral* veins are divided into an external and an internal group The internal group consists of 2 internal cerebral veins draining the deeper portions of the hemisphere, including the thalamus, the striate bodies, the corpus callosum, and the choroid plexuses. They unite to form the great vein of Galen which enters the junction of the inferior sagittal and the straight sinuses.

The external group includes the superior, inferior, and middle cerebral veins. The superior cerebral veins, 8 to 12 in number, empty into the sagittal sinus. The anterior veins run nearly at right angles, whereas the more posterior and larger veins are directed obliquely forward and medially The largest veins drain the precentral and postcentral areas in the middle and posterior third of the sagittal sinus. The middle cerebral vein is a large venous channel in the sylvian fissure which ends in the cavernous sinus. A vein communicating between the superior and middle cerebral veins, first described by Trolard, is known as the *great anastomotic vein of Trolard*. The inferior veins drain the undersurface of the hemisphere and join the cavernous the sphenoparietal, and the superior petrosal sinuses. The posterior anastomotic vein of Labbé forms a communication between the middle cerebral vein and the transverse sinus.

The *diploic* veins located in the cancellous layer between the outer and inner tables of the skull, are thin walled irregular, large vessels communicating with the dural sinuses and the meningeal veins intracranially and the veins of the epicranium extracranially The main branches are the frontal, the anterior temporal, the posterior temporal, and the occipital. The frontal diploic opens into the superior sagittal sinus and the supraorbital vein, the anterior temporal, into the sphenoparietal sinus and a deep temporal vein, the posterior temporal, into the transverse sinus the occipital into the occipital vein externally or into the confluence of sinuses.

The *emissary* veins are communicating channels between the intracranial venous sinuses and extracranial veins The two found most constantly are the parietal and the mastoid, connecting the sagittal and the transverse sinuses, respectively, with extracranial veins.

Dural Sinuses

The dural sinuses are endothelial-lined spaces between the periosteal and meningeal layers of the dura These channels convey the blood from the cerebral meningeal and the diploic veins into the general circulation They communicate with extracranial veins via emissary veins which run through foramens in the cranial bones There are 16 sinuses in all — 6 unpaired median sinuses and 5 pairs of lateral sinuses, the superior and inferior sagittal sinuses, the straight sinus, 2 transverse (lateral) sinuses, the occipital sinus, 2 superior and 2 inferior petrosal sinuses, 2 cavernous sinuses, 2 intercavernous sinuses, the basilar sinus or plexus, and the sphenoparietal sinus

The *superior sagittal sinus* is triangular in cross section; it is located in the convex margin of the falx cerebri, extending from the foramen cecum of the frontal bone in front of the crista galli to the internal occipital protuberance posteriorly, where it enters the confluence of sinuses The posterior portion of the sinus is much larger than the anterior one. The anatomy traversed by the sinus varies considerably. Usually, the sinus enters the right transverse sinus Among the superior cerebral veins, those arising from the vicinity of the motor and sensory cortex enter the posterior half of the sinus There are at least 3 venous lacunas on each side of the sinus — a frontal, a large occipital, and a large parietal — which empty into the superior cerebral veins; these lacunas are not present at birth, but are fairly well developed by the age of 7 Arachnoid granulations are said to extend into these lacunas, permitting the reabsorption of the cerebrospinal fluid.

The *inferior sagittal sinus* is located in the inferior free margin of the falx cerebri. It extends backward until it ends in the straight sinus which is located in the folds of the tentorium cerebelli The *straight*

sinus in turn empties into the confluence of sinuses, usually into the left transverse sinus. The straight sinus, which receives the great vein of Galen in addition to the inferior sagittal sinus, and several superior cerebellar veins on its inferior surface, is an important structure.

The *cavernous sinuses* on both sides of the body of the sphenoid, are traversed by many trabeculas. The sinuses communicate with each other via anterior and posterior communications. The oculomotor and trochlear nerves, and the ophthalmic branch of the trigeminal nerve, pass through the lateral sinus wall, and the abducens nerve and the carotid artery medially The cavernous sinuses communicate with the transverse sinuses through the superior and inferior petrosal sinuses and the basilar sinus, and with the pharyngeal and pterygoid plexuses via veins passing through the foramens ovale, spinosum, rotundum, and lacerum Of the veins emptying into the cavernous sinuses, the more important are the ophthalmic vein and the veins from the inferior surfaces of the frontal lobes in some, these veins, 3 or 4 in number first empty into the sphenoparietal sinuses, which in turn empty into the cavernous sinuses. In about 30 per cent of cases that come to operation, these large veins are present they are easily torn in exploration of the pituitary region and the uncal area of the temporal lobe.

The *transverse sinuses* are continuations of the superior or inferior sagittal sinuses, or of both, and of the straight sinus, the right transverse sinus is usually a continuation of the superior sagittal sinus whereas the left transverse is a continuation of the straight sinus. The sinuses extend laterally, forming an *S* curve in the mastoid area which extends into the jugular foramen as the jugular bulb and the internal jugular vein. The impressions of the transverse sinuses can be seen on the internal aspect of the skull extending laterally from the internal occipital protuberance toward the petro-occipital junction at which point the sinuses receive the superior petrosal vein, veins from the medulla and brain stem, and tributary veins from the superior surface of the cerebellum and the inferior surface of the temporal

and occipital lobes The temporal and occipital tributaries are frequently encountered in supratentorial exploration of the temporal lobe and the temporo-occipital cerebral junction

The *superior petrosal sinuses* arise from the cavernous sinuses, and are located in the anterior and lateral margins of the tentorium cerebelli attached to the superior edge of the petrous bone. In 50 per cent of the cases that come to operation the sinus passes over the root of the trigeminal nerve, and under the root of the nerve in the remainder; in some, it divides so that one portion of the sinus passes over the root and the other portion under the root of the gasserian ganglion

The *inferior petrosal sinuses* also arise in the cavernous sinuses In the region of the petro-occipital suture in the posterior fossa, the sinuses extend into the jugular foramen and empty into the jugular veins In their lateral course from the cavernous sinus they pass below the petrosphenoid ligament, together with the abducens nerve, and enter the jugular foramen between the glossopharyngeal and vagus nerves

Craniospinal Junction

The rotating atlas permits effortless turning of the head through 180° Vertical movements, which are less essential, are restricted by the internal and external craniocervical ligaments, which protect the bony structure from dislocation Short, cruciform ligaments maintain fixation of the odontoid processes Short ligaments also preserve the atlanto-axial relationship The articular capsule about each articular facet provides structural stability. Anteriorly, the anterior longitudinal ligament checks extension; posteriorly, the posterior longitudinal ligament, the ligamentum nuchae, and the supraspinous ligaments give security in hyperflexion.

The vertebral arteries, in their course through the transverse foramens, are well protected by bone, the lateral processes, however, are thin, and may be fractured The foramen magnum and the cer-

vical canal allow ample room for the upper spinal cord thus providing an extra margin of safety against injury

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and occipital lobes The temporal and occipital tributaries are frequently encountered in supratentorial exploration of the temporal lobe and the temporo-occipital cerebral junction

The *superior petrosal sinuses* arise from the cavernous sinuses, and are located in the anterior and lateral margins of the tentorium cerebelli attached to the superior edge of the petrous bone In 50 per cent of the cases that come to operation the sinus passes over the root of the trigeminal nerve, and under the root of the nerve in the remainder; in some, it divides so that one portion of the sinus passes over the root and the other portion under the root of the gasserian ganglion

The *inferior petrosal sinuses* also arise in the cavernous sinuses In the region of the petro-occipital suture in the posterior fossa, the sinuses extend into the jugular foramen and empty into the jugular veins In their lateral course from the cavernous sinus they pass below the petrosphenoid ligament, together with the abducens nerve, and enter the jugular foramen between the glossopharyngeal and vagus nerves

Craniospinal Junction

The rotating atlas permits effortless turning of the head through 180° Vertical movements, which are less essential, are restricted by the internal and external craniocervical ligaments, which protect the bony structure from dislocation Short, cruciform ligaments maintain fixation of the odontoid processes Short ligaments also preserve the atlanto-axial relationship The articular capsule about each articular facet provides structural stability Anteriorly, the anterior longitudinal ligament checks extension, posteriorly, the posterior longitudinal ligament, the ligamentum nuchae, and the supraspinous ligaments give security in hyperflexion

The vertebral arteries, in their course through the transverse foramens, are well protected by bone; the lateral processes, however, are thin, and may be fractured. The foramen magnum and the cer-

vical canal allow ample room for the upper spinal cord, thus providing an extra margin of safety against injury

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Chapter III

MECHANISMS OF HEAD INJURY

Most of the causes of head injuries fall into two main groups. direct and indirect A moving object striking a nonmoving or slower moving head is a direct cause of injury, whereas the sudden stopping or deceleration which occurs when the moving body and head come in contact with a nonmoving or slower moving object is an indirect cause Another indirect cause is the sudden setting into motion or acceleration of the body and the head at different rates of acceleration; this is a frequent cause of whiplash injuries of the head and neck When the cause is indirect, there is a likelihood that other body structures have also suffered injury Injuries at the craniospinal junction are particularly frequent, since a sudden acceleration or deceleration of the head may cause stress on the neck structures which fix the head to the body, if severe enough, the stress may injure ligaments and muscles or fracture the cervical spine and injure the spinal cord and its blood supply. A minor head injury may thus be accompanied by a major injury of the cervical spine Table 1 lists the causes in a series of 716 cases

Whether the cause of injury is direct or indirect, the physical effects may include deformation of the skull as a result of compression or in-

bending of the bone and/or acceleration or deceleration of the head and of the intracranial contents.

TABLE 1 *Causes of Head Injury in 716 Cases**

Cause	*Number of cases*	*Percentage*
Automobile accidents	146	20.4
Struck by vehicles	122	17.0
Blows	*251*	*35.1*
Object unknown	102	14.2
Fighting and beatings	70	9.7
Blunt objects	47	6.6
Metal objects	30	4.9
Collision in ball play	2	0.3
Falls	159	22.2
Accidents while drunk	31	4.3
Bicycle accidents	6	0.8
Attempted suicide	1	0.1

* Part of the series of 1,285 cases.

Collision between two objects creates energy. Whether the moving head hits a stationary object, or a moving object hits the stationary head, or head and object moving at different velocities hit each other, the head absorbs some of the energy of the collision, the amount depending upon the mass, density, shape, and relative velocity of the striking object. The damage which must result when sufficient energy is absorbed includes skull fracture with or without scalp injury, cerebral contusion or laceration, and concussion. Collision between the moving head and a stationary object decelerates the head and its contents; contact between a moving object and a head at rest but free to move accelerates the head and its contents linearly, angularly, or both; contact between a moving object and head not free to move, on the other hand, causes deformation primarily, with acceleration occurring only in the region of the deformation. These circumstances can be characterized as dynamic conditions. Under static conditions, such as occur

when the head is caught between a slowly moving heavy weight and a fixed object, there is deformation without acceleration or deceleration

The size and shape of the object with which the head comes in contact considerably affect the amount and character of the deformation and the occurrence of acceleration or deceleration. A small, sharp object, such as a stiletto, may penetrate the scalp, skull, and brain without causing appreciable deformation or acceleration and without producing unconsciousness Small, light objects traveling at high speeds may cause little deformation or acceleration when they strike the head because the kinetic energy is slight On the other hand, a large mass moving at a low speed may produce considerable deformation without appreciable acceleration or deceleration

The physical effects of blows to the head are (*1*) deformation of the skull, with possible fracture or squeezing of the intracranial contents, or both, due to a decrease in volume, (2) sudden increase in intracranial pressure at the time of impact, (*3*) mass movements of the intracranial contents due to different acceleration rates of the skull and its contents, (*4*) distortion of the skull and dural septums, (*5*) shearing off of a portion of the head without necessarily producing an appreciable increase in intracranial pressure, (*6*) shearing and tearing of intracranial contents due to greatly increased intracranial pressure, as in bullet and shell fragment injuries, (*7*) combinations of any of the above effects

These various effects may cause injury by one of three mechanisms: (*1*) by compressing, pushing together, or "squeezing" the tissues, (2) by tension, or by tearing the tissues apart, (*3*) by shearing, or sliding of tissues over each other All three of these mechanisms may operate simultaneously in different areas or in different directions in the same area For instance, the scalp may be compressed or mashed at the site of the blow; the skull may be crushed by the compressive forces or a fracture may occur due to tensile stresses, if rotation of the head occurs, portions of the brain may be compressed by contact with adjacent bone; other portions may be torn by the tension produced as the brain rotates

with respect to the skull, finally, shearing may occur due to pressure gradients, principally in the region of the brain stem.

The role of sudden compression of the chest and abdomen in the causation of brain injury is not well understood Underwater blasts may cause intracranial damage through pressure impulses along the vascular channels. In the monkey the injection of 2 to 4 cc. of fluid into the carotid artery results in an expansion of the pial arteries of 50 to 250 μ in size. Sudden compression of the chest by hand also causes expansion of the vessels, particularly the pial veins. The presence of subcortical areas of damage described by Strich may be more easily explained on the basis of injury by thoracic compression at the time of impact, leading to petechial hemorrhages in the white matter along the venous channels (*see* p 305)

Many physical and chemical changes, including hemorrhage or massive bleeding swelling and edema of the tissues, and the secondary pathologic phenomena of absorption repair or inflammation, occur after the initial effects of the blow In serious injuries, there may be hemorrhagic extravasions in the brain stem secondary to herniation of the uncus and brain stem as a result of mass lesions and cerebral swelling

Scalp Injuries

The type of scalp wound found in head injury is usually related to the physical properties of the injuring object. A blow by a blunt object may or may not produce a readily visible scalp bruise, although close examination will often reveal an area of contusion A sharp object will puncture or lacerate the scalp and if it is moving rapidly may cause extensive contusion around the central punctured area. A thin sharp, pointed instrument (icepick, stiletto) may cut or puncture the scalp only slightly but the intracranial damage may be much more serious. A hard blow by a heavy unyielding object (blackjack, steel bar, heavy

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tools, steel handles) not only bruises but also lacerates the scalp The scalp injuries incurred in automobile accidents may be (*1*) lacerations, (2) cuts by broken glass, (*3*) contusions from contact with blunt and unyielding structures, and (*4*) evulsions, particularly if the body has been dragged by the vehicle

Varied scalp wounds are caused by shot, bullets, or shell fragments Shot cause multiple small wounds which become ecchymotic, and often the pellets are palpable in the scalp, if the discharge is at close range, so that the impact is at or near muzzle velocity, the injury is usually devastating and may be fatal The entrance wound of a bullet is usually small, with or without a surrounding area of burned skin, if there is an exit wound, it is much larger and the intracranial damage at the exit is apt to be extensive. Shell fragments lacerate, evulse, or destroy small or large areas of the scalp Less tissue is destroyed by low-velocity than by high-velocity missiles While the edges of a wound caused by a low-velocity missile are ragged and contaminated, the high-velocity missile devitalizes the tissue for as much as 2 to 3 cm around the actual wound

Skull Fracture

The deformations of the skull resulting from a variety of causes have been studied by the stresscoat and strain gage technics The stresscoat technic consists of applying a coat of brittle lacquer (the stresscoat) to a test object, in this case the skull When a certain amount of strain occurs in the underlying material, the lacquer cracks, and the pattern of the cracks reveals the extent of the deformation which the blow produced in the object An electric strain gage permits exact measurement of the amount of strain applied as well as its duration

Experiments were carried out on the calvariums of dogs and monkeys under anesthesia, on the calvariums of dead animals with contents intact and on the dry skulls, and on human cadaver skulls with contents intact and on dry skulls The dry cadaver skulls were stress-



Fig. 5 Stresscoat patterns after blows to skulls. Posteroparietal blow to skull of anesthetized dog (a^1) to skull with intact contents (a^2) and to dry skull (a^3) Midparietofrontal blow to skull of anesthetized rhesus monkey (b^1) to skull with intact contents (b^2) and to dry skull (b^3) Posteroparietal hammer blow to human cadaver skull (c^1) and impact of deceleration on dry skull (c^2) Laterofrontal hammer blow to human cadaver skull (d^1), and impact of deceleration on dry skull (d^2)

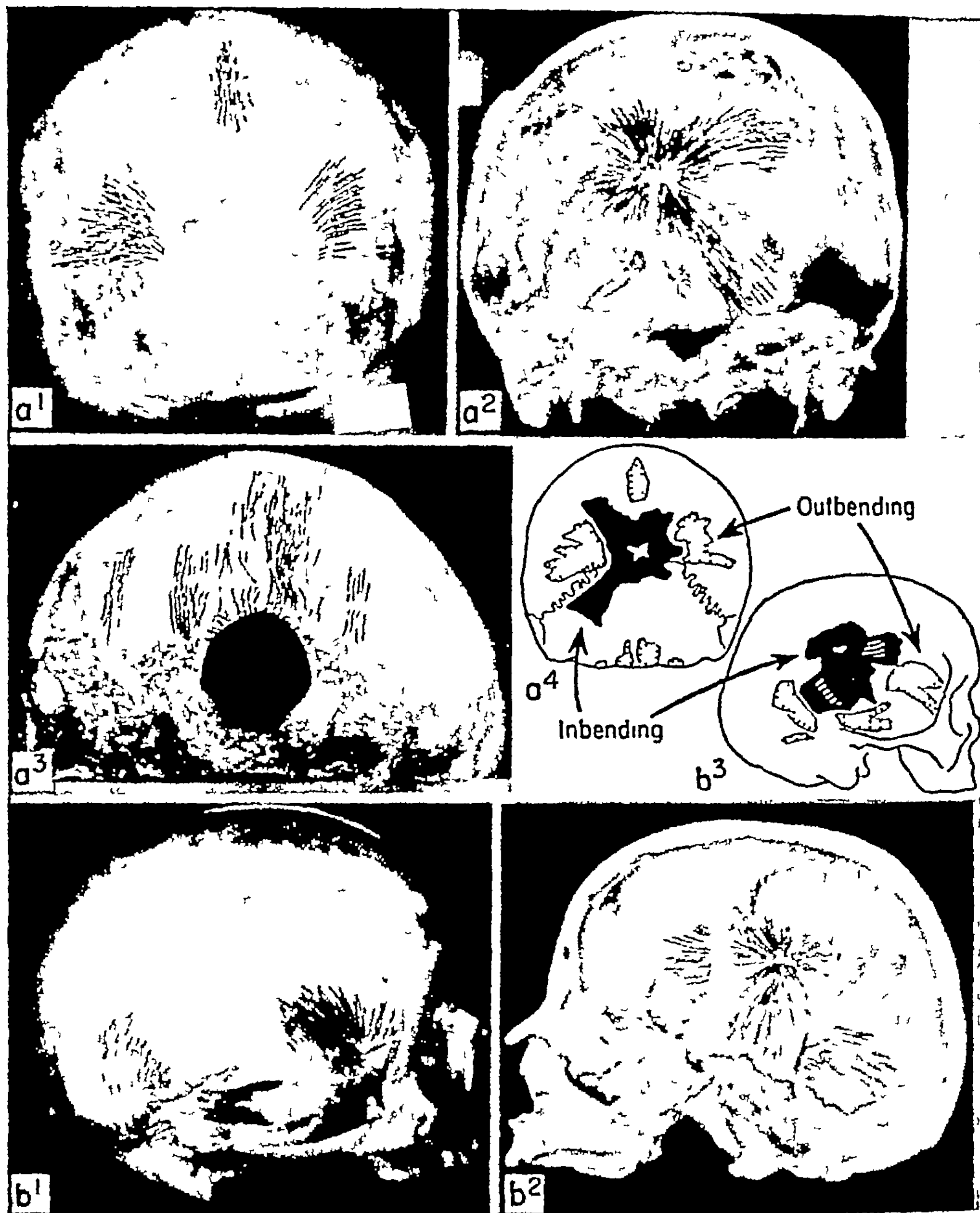


Fig 6 Stresscoat patterns after low-velocity blows (dry human skulls were dropped onto a flat steel surface) (*a*) Midoccipital blow, outer cracks from outbending of bone (a^1), inner cracks from inbending of bone (a^2), cracks at base near foramen magnum from tearing-apart stresses (a^3), and composite diagram of inbending (black) and outbending (dotted) (a^4). (*b*) Parietal blow, outer cracks from tearing-apart stresses in laterofrontal, temporal, and posteroparietal regions (b^1), inner cracks from inbending (b^2), and composite diagram of inbending (black) and outbending (dotted) (b^3).

coated on both inner and outer surfaces in order to study the strain patterns after a blow, due to inbending and outbending of bone. The cracks in the stresscoat on the outer surface revealed the stresses due to outbending, those on the inner surface, the stresses due to inbending

Figures 5 and 6 illustrate some of the results of our studies While the degree of skull deformation in anesthetized animals and of their dry skulls differed somewhat, there was considerable correlation both in the direction and the pattern of the cracks in the stresscoat. The same was found to be true of the human skulls. A midoccipital blow caused an irregular area of inbending at the point of impact, with outbended areas about the foramen magnum, in the parieto-occipital region bilaterally, and in the interparietal midline area anteriorly (Fig 6) The most severe stresses occurred in the occipital squama With a parietal blow (Figs. 7-9), the area at the point of impact bent in, with outbent areas around it, the most severe outbending occurred in the anterior temporal area.

From the test results we concluded that a skull which strikes, or is struck by, a smooth flat surface at moderate velocities (as in falls, industrial accidents, and automobile accidents at low speeds) simultaneously bends inward at the point of impact and outward at some distance from it. A stronger blow causes greater deformation, several sites around the inbended area bend outward, producing an undulating pattern of in and outbending At higher than moderate velocity, a blow causes a localized injury — a depressed or perforated fracture. Missiles of extremely high velocity, *e g* high-speed rifle bullets, may shatter the entire skull by (*1*) transmission of the missile's high kinetic energy, and (2) the shattering effect of the bullet's passage through the skull.

Experiments on the dry skull are illustrated in Figure 10, on impact, a ¼ inch solid steel pellet traveling at 50 feet per second caused a linear fracture, the same pellet traveling at 90 feet per second neatly perforated the skull

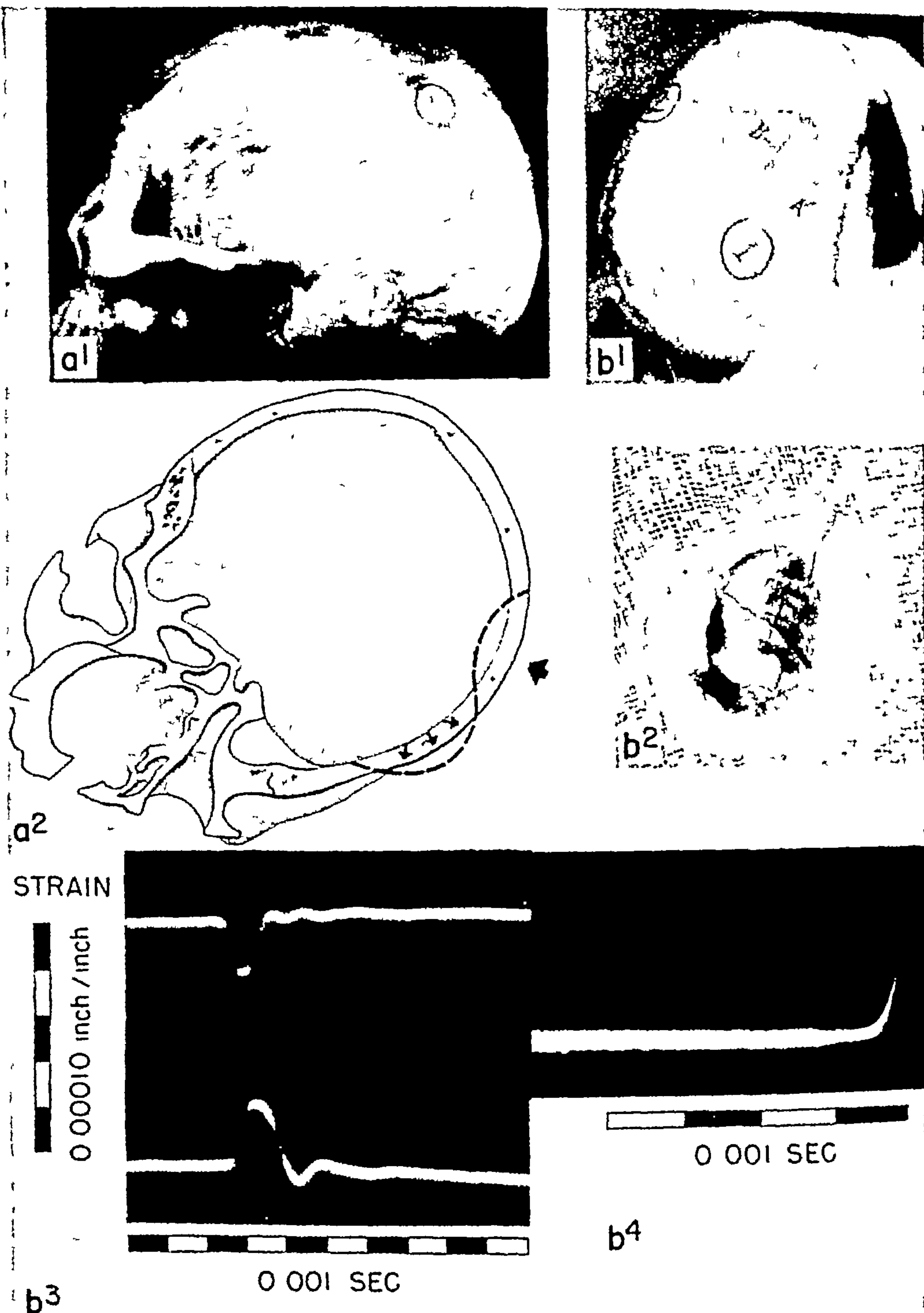


Fig 7. Parietal blows (circle) to cadaver skull. (*a*) Cracks in stresscoat in temporal region (*a*[1]), and diagram of deformation pattern (inbending at point of contact, outbending elsewhere) (*a*[2]) (*b*) Strain gage studies 4 hours after death: (*b*[1]) skull prepared for study, (*b*[2]) strain gage torn by fracture, (*b*[3]) oscillogram showing total time (0 004 sec.) of skull deformation, (*b*[4]) oscillogram with one beam of oscillograph recording contact between striking object and scalp while other beam continues recording until circuit is opened by fracture line, scalp compression in 0 0006 second skull deformation and fracture 0 0006 second later.

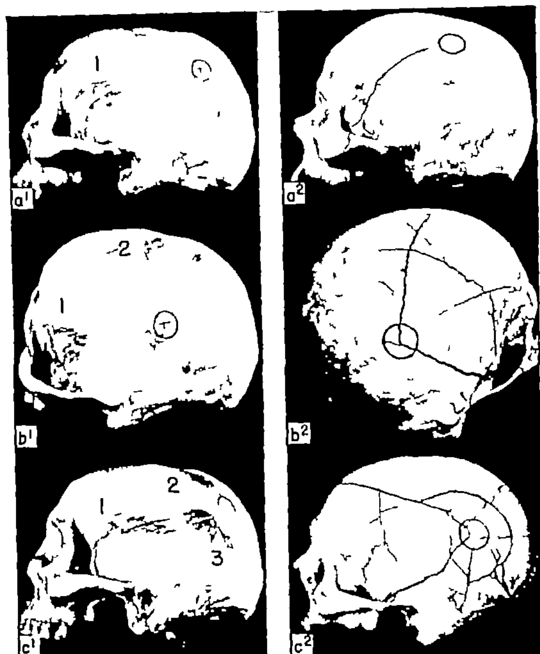


Fig. 8. Posteroparietal blows to cadaver skulls. (a^1) Primary stress (1) in temporal area from outbending; (a^2) moderate blow with single linear fracture. (b^1) Heavier blow with primary (1) and secondary (2) stresses, and (b^2) moderately heavy blow with triple linear fracture. (c^1) Severe blow, with areas of primary (1), secondary (2), and tertiary (3) stresses. (c^2) Severe blow with multilinear fracture, comminution, roughly following inbent area, and depression at point of impact.

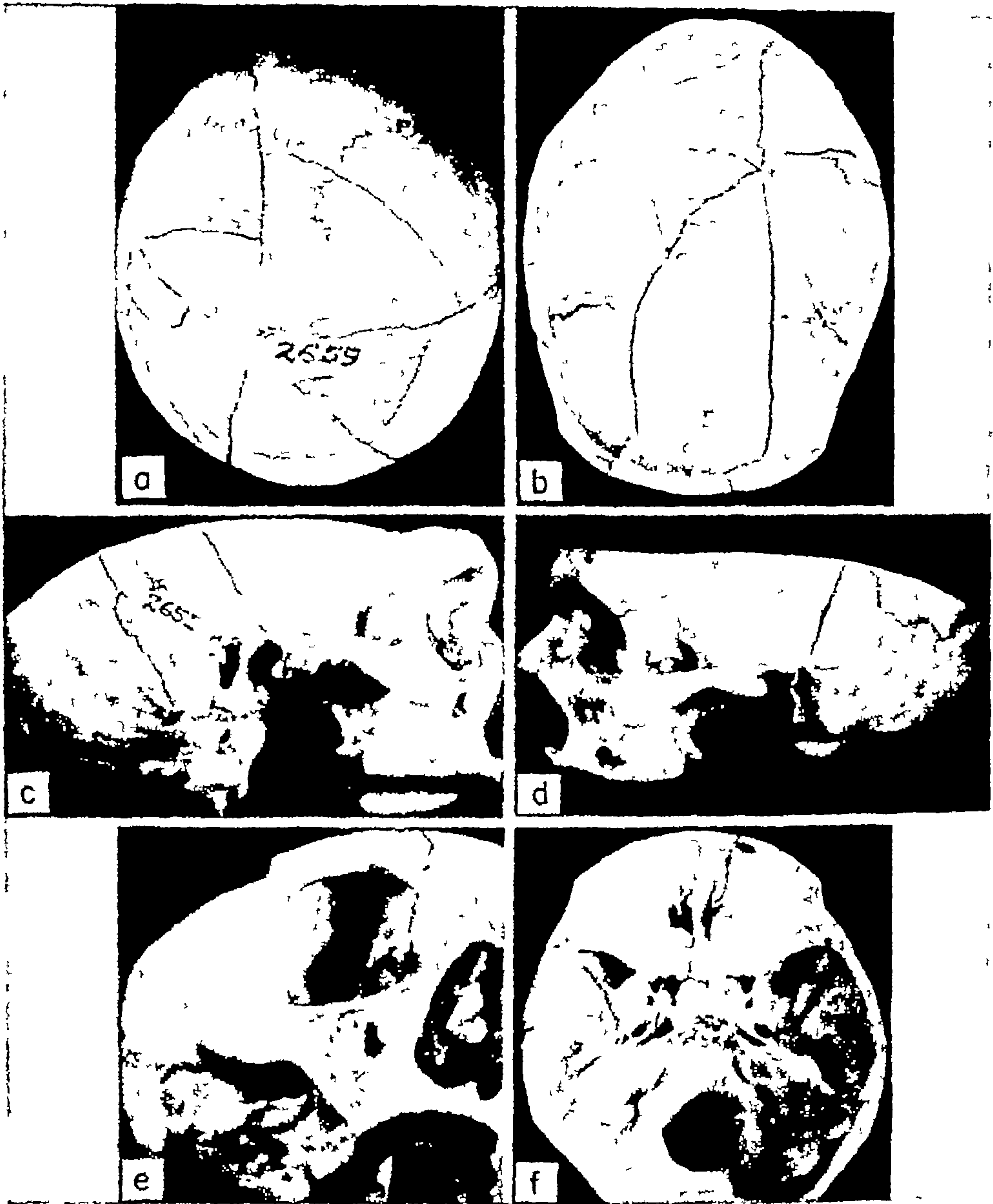


Fig. 9. Interparietal blow with stellate fracture in cadaver skull. (*a*) Separate fracture lines in outer table. (*b*) Fracture lines in inner table. Fractures extending to temporal and mastoid regions on right (*c*) and into external auditory meatus on left (*d*). (*e–f*) Fracture extending into orbital roof, involving cribriform plate, dorsal surface of right petrous bone, and temporosphenoid junction of right middle fossa

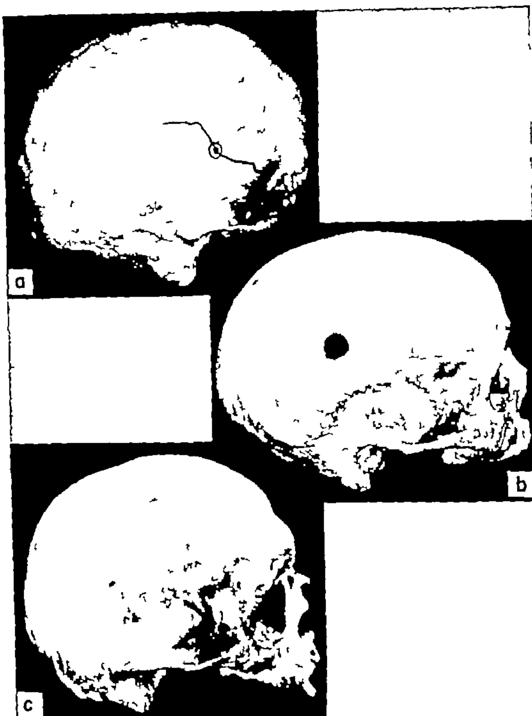


Fig 10 Relation of velocity and size of object to type of fracture. (*a*) Steel ball, ¾ inch diameter impact velocity 50 feet per second linear fracture. (*b*) Same ball impact velocity 90 feet per second clear perforation. (*c*) Steel ball ½ inch diameter impact velocity 90 feet per second outer table indented, smaller object at same velocity lacked energy to perforate skull.

Linear Fractures

Analysis of the stresscoat patterns reveals that a low-velocity blow causes inbending of the area of impact and outbending of the adjacent bone A linear fracture begins in the outbended area and extends both toward and away from the point of impact The extension toward the impact area can be explained by the fact that after initial inbending the bone bends outward and thus becomes a region of stress concentration This may not be manifest in every instance, due to anatomic variations in the thickness and stress characteristics of the skull. A stronger blow, transmitting greater energy, causes a comminuted or multilinear fracture, also beginning in the outbended areas and extending toward and away from the point of impact With blows of still greater energy, the bone breaks circumferentially about the point of impact, the extent of the fracture being limited by the previously formed radial fracture lines The deformation and fracture pattern is governed to some extent by the properties of the object striking or being struck. Thus, if the head strikes a yielding object, such as a flat, thin, metal sheet, the skull will be only slightly deformed, with a corresponding decrease in the likelihood of fracture.

With blows in the parietal region (Figs 7-9), the primary stress area, and therefore the site for the start of a linear fracture, is the anterior temporal region With increasing energy, additional cracks appear in the stresscoat, indicating, in the order of their appearance, the areas of secondary and tertiary stresses Experiments further showed that approximately 0 0012 second is required to produce a fracture; during the first half of this interval, the scalp is deformed and during the second, the bone undergoes deformation u appears Blows delivered to the midfrontal, anteri midoccipital, and right and left posterior parietal r cadaver skulls showed that the presence of hair, scalp, contents had a negligible effect on the posit

but not on the energy required to produce them. The energy ranged from 425 lb per square inch to over 900 lb (Table 2) Studies revealed the scalp s tremendous capacity to absorb energy, at least ten times as much energy was required to fracture an intact cadaver skull as a dry skull, for which as little as 40 lb per square inch sufficed The average tensile strength of compact bone in the cadaver skull has been found to be 10,150 psi, the average compressive strength of compact bone at right angles to its surface, about 24,500 psi, and parallel to its surface about 23,100 psi.[38] While the energy requirements for fractures of the embalmed cadaver heads and those of living individuals may differ, we believe that this factor is of no greater significance than are the normal individual differences in the heads of living persons.

TABLE 2. *Experimental Skull Fracture in Cadaver Heads**

Head no	*Head weight lb*	*Distance dropped in*	*Total energy in-lb*	*Accelera tion g*	*Time sec*	*Site of blow†*	*Site of fracture†*
1	8.56	60	515	240	0.0013	R O	O to P
2	10 44	60	629	460	0 0013	L.F	L.F to L.P
3	6.81	71.5	489	372	0.0015	R.F	R.F to L.P
4	9.25	54	500	456	0.0011	L.O	None
5	9.50	65	618	557	0.0019	L.O	None

* Head no 3 Negro male· others, white male.
† R., right, O., occipital L., left F., frontal P., petrous

Figure 11 shows the results of tests to determine the areas of greatest weakness (primary stress areas) of the human skull. Each of 100 skulls was divided into 12 quadrangular areas and each area was subjected to a blow with deceleration. The results lead us to believe that the location of a linear fracture can be predicted with reasonable accuracy if the site of impact is known and conversely, that the point of impact can be determined from the location of the fracture (Fig 12)

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but not on the energy required to produce them. The energy ranged from 425 lb. per square inch to over 900 lb (Table 2) Studies revealed the scalp's tremendous capacity to absorb energy, at least ten times as much energy was required to fracture an intact cadaver skull as a dry skull, for which as little as 40 lb per square inch sufficed The average tensile strength of compact bone in the cadaver skull has been found to be 10,150 psi, the average compressive strength of compact bone at right angles to its surface, about 24,500 psi, and parallel to its surface about 23,100 psi [28] While the energy requirements for fractures of the embalmed cadaver heads and those of living individuals may differ, we believe that this factor is of no greater significance than are the normal individual differences in the heads of living persons.

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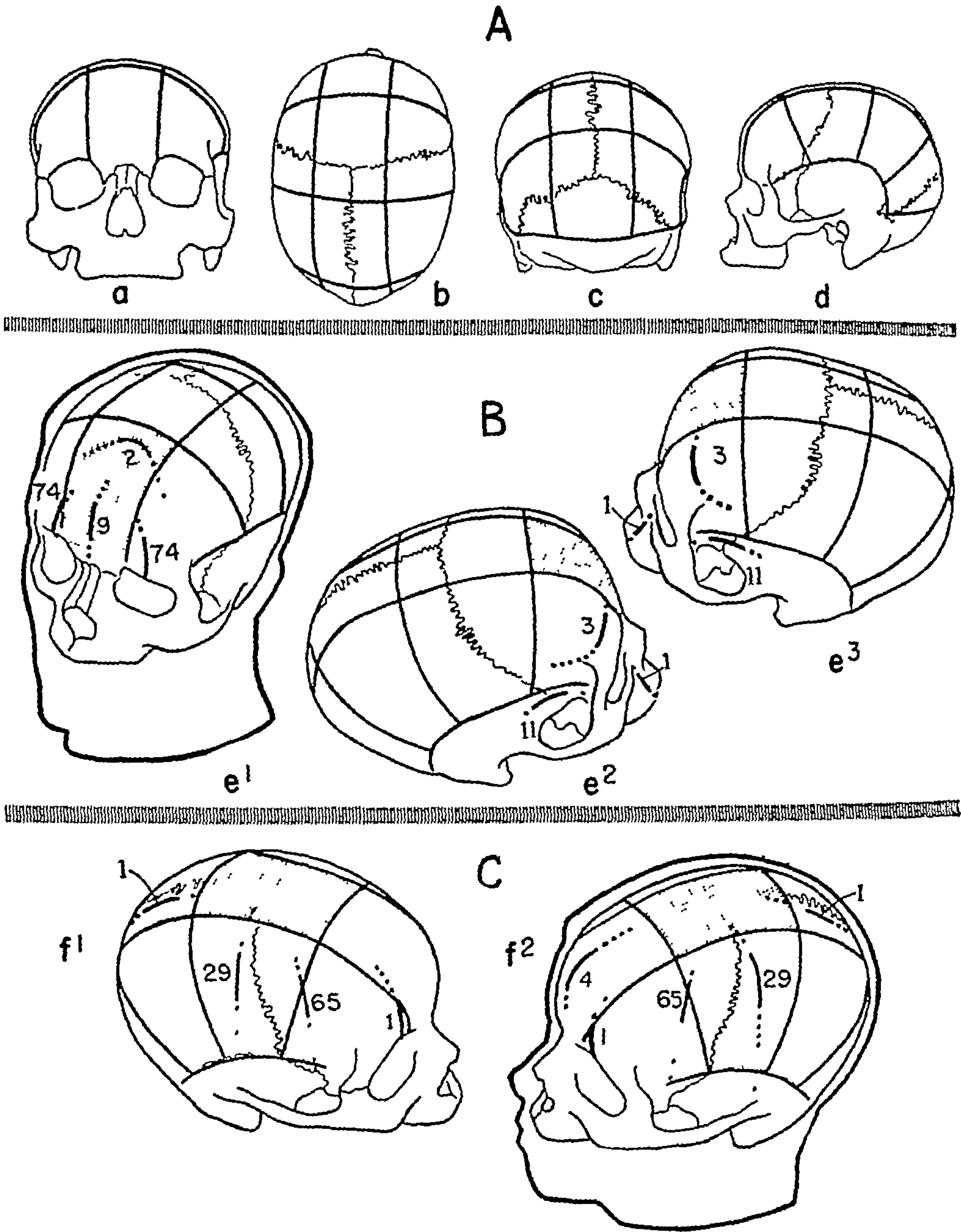


Fig 11. Sites of fracture from low-velocity blows to 100 cadaver skulls, each divided (*A*) into rectangular areas of about $2 \times 1\frac{1}{2}$ inches Dotted areas show sites of blows, numbers and lines show the percentages and directions of fractures in each area (*B*) Midfrontal blows (*C*) Anterointerparietal blows (*D*) Left anteroparietal blows (*E*) Left posteroparietal blows (*F*) Left parieto-occipital blows (*G*) Posterointerparietal blows (*H*) Midoccipital blows (*I*) Left frontal blows

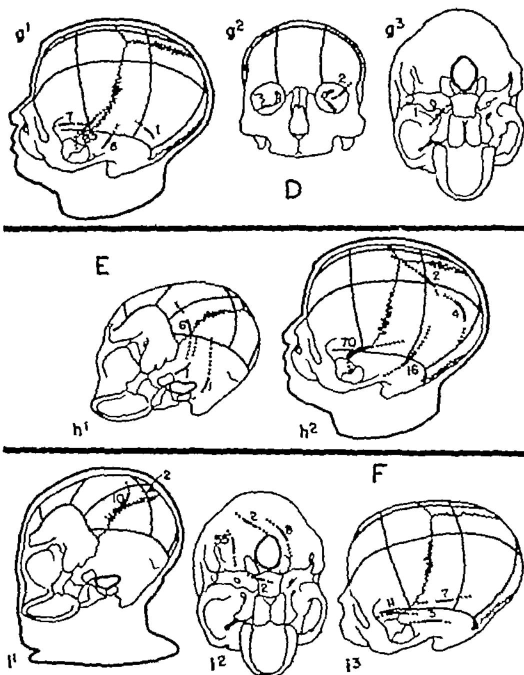


Fig 11 (*continued*) *See* legend page 72.

poral area (Fig 7) With blows to the anterior portions of the head, linear fractures may involve the roof of the orbit on one or both sides Often, the cribriform plate is involved, and the fracture line may extend laterally or through the pituitary fossa into the basilar process of

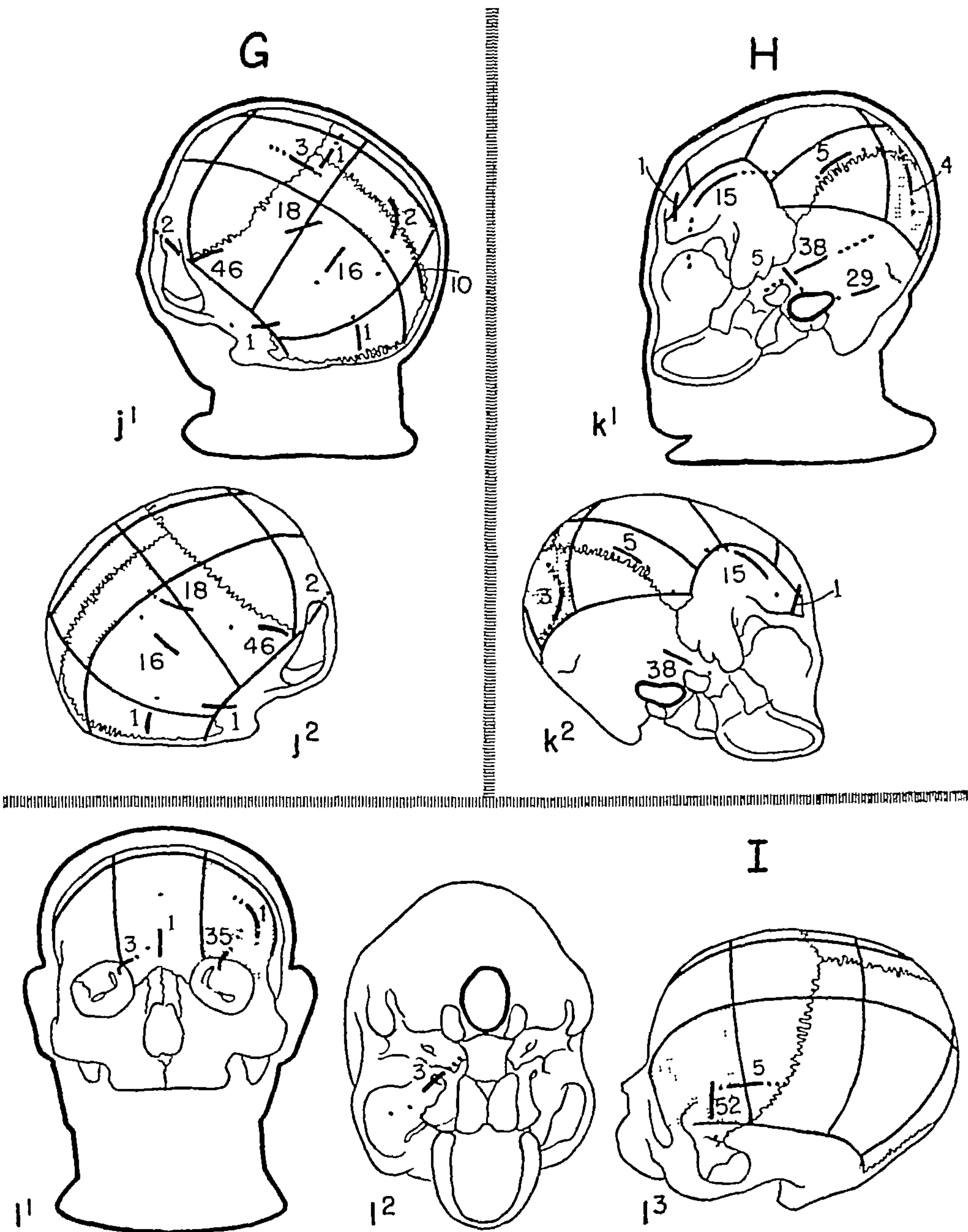


Fig 11 (*continued*) *See* legend page 72

the occipital bone Blows in the interparietal regions cause linear fractures which extend perpendicularly toward the base, involve the lesser wing of the sphenoid, and possibly the pituitary fossa or the sphenoid sinus, or both Posterior interparietal blows may cause fractures extend-

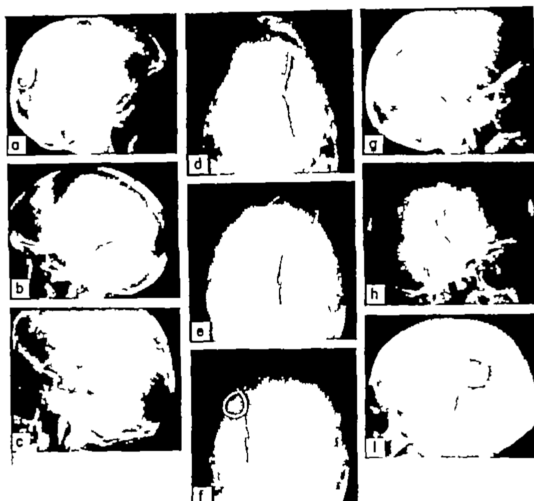


Fig 12 Linear skull fractures area of impact (radiopaque wire circle) known from presence of scalp laceration or contusion note increasing width of fracture with distance from impact point. (*a–c*) Posteroparietal blows near midline, with fractures extending downward and forward toward temporal area (*d–f*) Occipital blows, with fractures extending lateral to or into foramen magnum (*g–i*) Inferoparietal blows, with fractures extending perpendicularly forward and downward toward temporal area.

ing toward the posterior base and petrous bone and involve the base from side to side. Blows to the back of the head may cause fractures which extend into the foramen magnum or to the lateral aspect of the foramen into the jugular foramen, in some instances, these fractures may cross the petrous bone into the middle fossa.

The midline frontal, midline occipital, paired frontosphenoid, and parietopetrosal buttresses (*see* p. 38) may govern, more or less, the

direction of linear fractures resulting from blows upon the vault, although in our studies we have not found this to be so invariably. The direction of the fracture in relation to the buttresses, however, is important, if it is at right angles to the buttress, the fracture line will extend through it A fracture extending toward the base from an interparietal blow can bisect the petrous bone lengthwise Other fractures can cross the midline and extend through the middle of the frontal bone toward the base of the skull The midline occipital area is also readily fractured

Fractures of Outer and Inner Tables (Figs. 13-14)

In the normal skull, the outer table is thick, the inner table thin; a blow which fractures the outer table commonly also fractures the inner table Occasionally, however, the blow may be just of sufficient energy to fracture the outer table but only deform the inner table. When the inner table is thicker than the outer table (*see* p 37), however, the former only may fracture In fractures involving the base, the line of fracture may extend toward the area of impact and involve the inner table only (Fig 14)

Sutural separations result from tearing-apart forces due to outbending of bone around the area of inbending at the site of the blow (*see* Fig 16)

Basal Fractures (Fig. 15)

In 6 per cent of the skulls, discrete stress patterns developed at the base which were not associated with stress lines in the calvarium Since the base is perforated by many foramens, which are areas of stress concentration, it may well be the explanation for such basal fractures In general, however, basal fractures are extensions of fractures of the vault. Blows to the frontal and occipital areas produce side-to-side stresses which cause basal fractures extending anteroposteriorly or posteroanteriorly, blows to the interparietal and midfrontal areas produce tearing-apart forces in the anteroposterior direction, so that the basal frac-

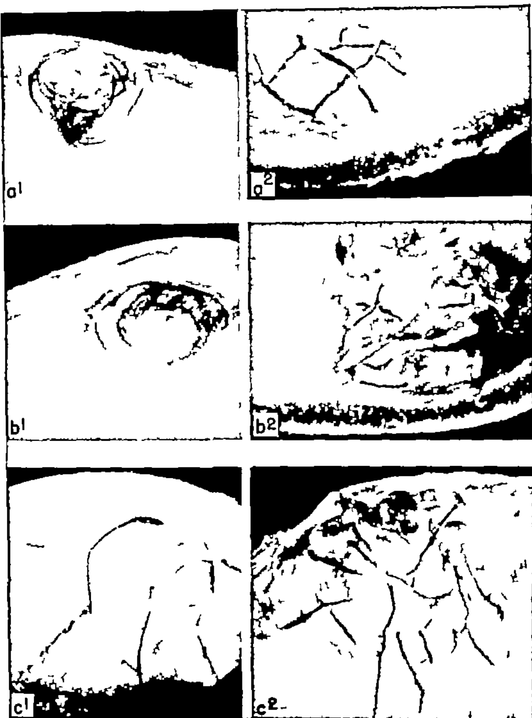


Fig 13 Depressed fractures in cadaver skull (scalp and contents intact) by hammer blows. (a^1–c^1) External configuration. (a^2–c^2) Inner table fragmentation note more extensive fragmentation of inner table.

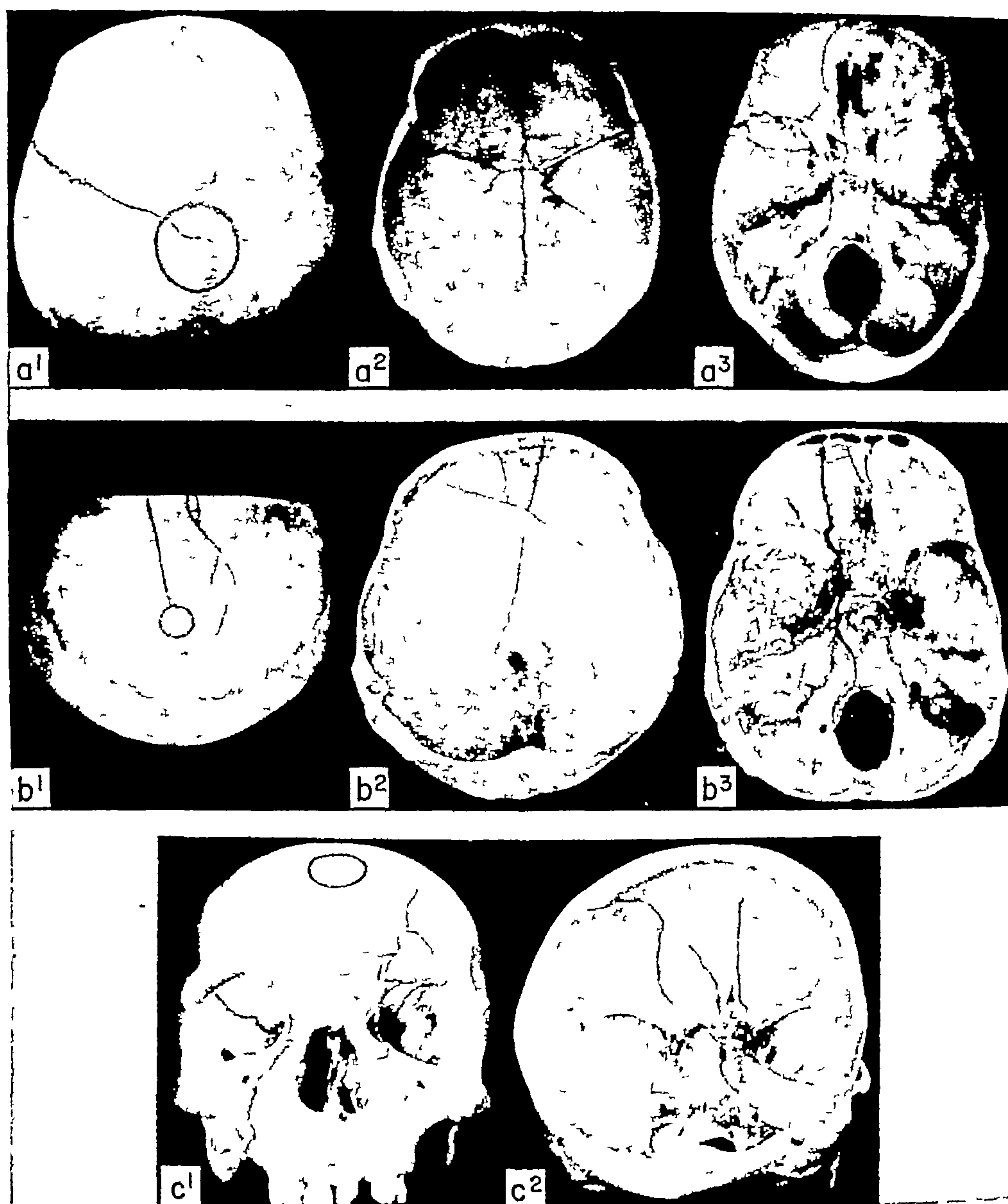


Fig 14 Fractures in cadaver skulls (*a*) Interparietal blow (circle), with extensive fracture of inner table (a^2) and basal involvement (a^3) (*b*) Frontal blow (circle), with fracture extending toward impact area, involving outer (b^2) and inner (b^3) tables, and extending through cribriform plate into cavernous sinus area with involvement of occipital basilar process (*c*) Frontal blow (circle), with triangular fracture involving orbital roof (c^1) and unrelated fracture of right side of cribriform plate (c^2)

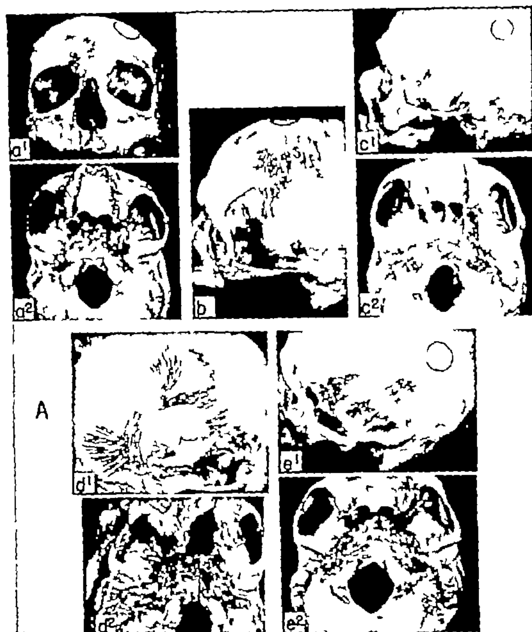


Fig 15 Basilar stresses with blows in various regions. (*A*) Left frontal blow (*a*) stresses at orbital roofs and base. Parietofrontal blow at midline (*b*) stresses above external acoustic meatus and lateral frontal and temporal bones. Left parietal blow (*c*) stresses at superior border of external acoustic meatus and base. Low temporoparietal blow (*d*) transverse stresses at base. Posteroparietal blow (*e*), anteroposterior stresses at base. (***Continued on next page***)

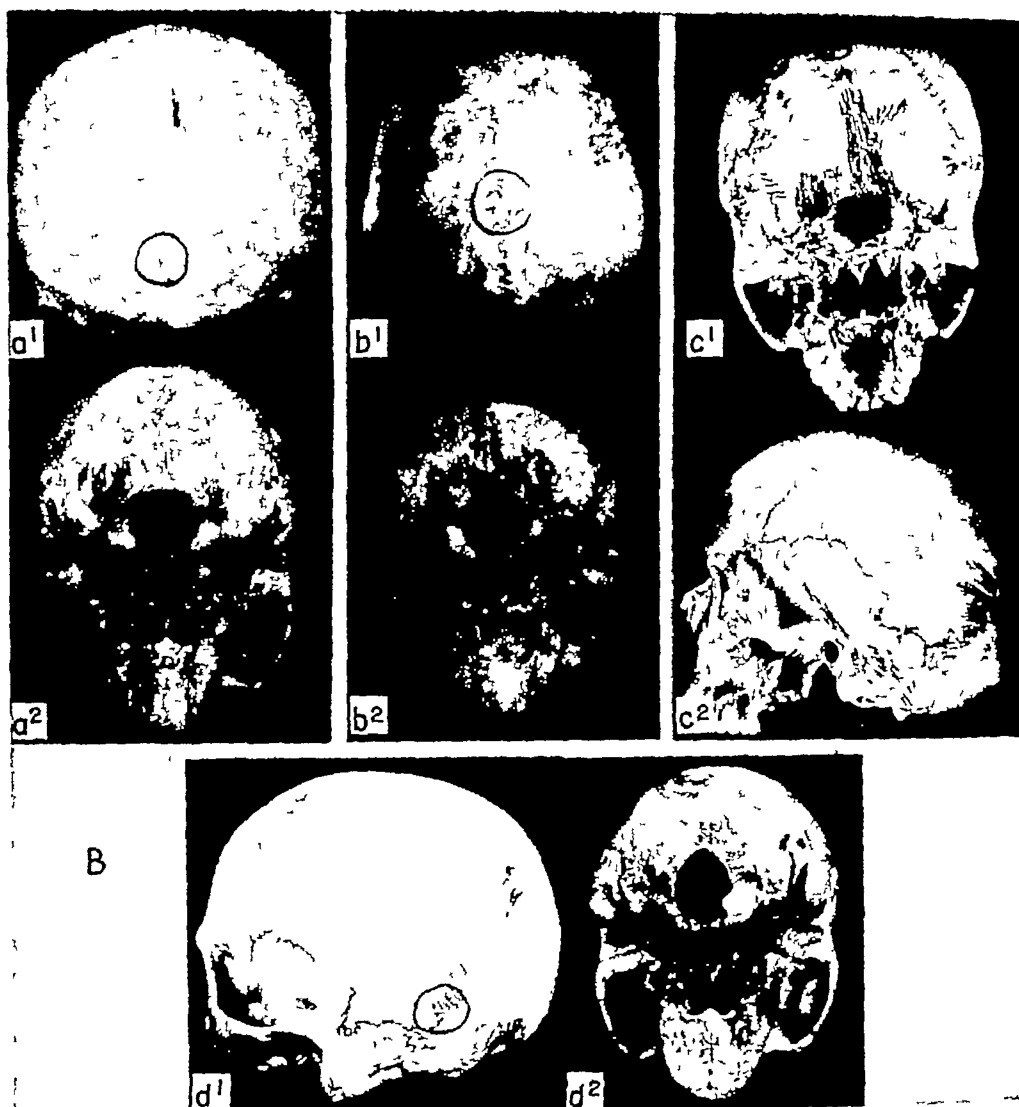


Fig 15 (*continued*) (*B*) Midoccipital blow (*a*), transverse stresses at base Interparietal blow (*b*), stresses in right parietotemporal area and base Midoccipital blow to left of midline (*c*), transverse stresses at base Parieto-occipital blow near left mastoid area (*d*), oblique stresses across base.

tures are transverse. Stresscoat studies showed that 4 to 5 per cent of blows to the vault in the frontal and interparietal areas produced discrete basal stresses, and blows to the more posterior portions of the head produced transverse stresses about the foramen magnum (Fig 11).

Curvilinear fractures about the foramen magnum were noted as early as 1873,[10] and have continued to be described in the literature.[8, 10]

They may occur from indirect blows, e.g in falls in which the vertebral column is pushed toward the cranial cavity, or by blows which push the head down upon the vertebral column The fracture lines may surround the foramen magnum circularly, or, more commonly, be limited to one or more short lateral cracks along the border of the fora men Blows to the lower jaw which push the condyloid processes toward the base may involve the middle fossa, in one such case, de pression of both condyloid fossae was found

In summary, it may be said that fractures of the base may be (*1*) extensions of vault fractures, (2) basal fractures unassociated with vault fractures, (*3*) due to impact on the lower jaw, (*4*) due to indirect forces, and (*5*) direct blows along the circumference at the base, acting like a hatchet splitting through the grain of a piece of wood.[47]

Depressed Fractures

There are seven varieties of depressed fractures, depending upon the velocity, the kinetic energy, and to some extent on the shape of the object (*see* Fig *10*)

(*1*) *Perforating Fractures*

An object moving at high velocity, *e g* , a rifle bullet, perforates the skull and shatters the bone by the energy which it imparts to the bone in its passage through the skull (*see* Fig 51*B*) The tremendously in creased intracranial pressure at the time of the passage of the mis sile through the head may also be a factor in the shattering of the bone.

(2) *Penetrating Fractures*

An object moving at fairly high velocity, *e g* a shell fragment, pene trates the skull and drives bone fragments into the brain However, if the kinetic energy is low and most of it is dissipated upon impact with the bone, only the outer table may be depressed or penetrated

(3) Depressed Fracture with Radial Linear Fractures and Separation of Tables

A blunt object, *e.g.*, a baseball, brick, or hammer, moving at moderate velocity, will cause a depression, but most of the kinetic energy may be absorbed in this process. The bone is fragmented by several radial fractures, the border of the depressed area presenting a curvilinear fracture, in some cases involving only the outer table, caused by tearing-apart forces on the external surface of the skull at the junction of inbended and normal bone. The inner table is usually more fragmented than the outer one, and there is greater ramification of the fracture lines. If the kinetic energy is almost completely dissipated at impact, only the outer table may suffer damage. On the other hand, if the inner table is thicker than the outer, the former may be fractured despite minimal depression of the outer table (Fig. 13).

(4) Depressed Fracture with Deformation Elsewhere in Skull

A slowly moving object delivering a localized blow to the skull will cause a depression at the point of impact and an outbending in other regions, so that 1 or 2 linear fractures resulting from the outbending may extend toward the area of impact (Fig. 16).

(5) Depressed Fracture Patterned after Striking Object

A fairly sharp or pointed object, moving rather rapidly, may cause a depression shaped more or less like the object. Such a fracture may or may not be accompanied by linear fractures in the neighboring bone.

(6) Depressed, Comminuted Fracture

A slowly moving blunt object of high kinetic energy, such as encountered in slow deceleration accidents, may produce extensive comminution, with radial fracture lines extending from the center of the area of impact and circular fracture lines at varying distances around the area. The circular fracture lines are caused by the increasing de-



Fig 16. Depressed fractures. (*a–b*) Midline posteroparietal fracture, with extensive separation of right lambdoid suture. (*c*) Midline frontal depressed fracture associated with linear fractures extending toward depression

pression of bone. The radius of the skull's curvature in the impact area influences these successive lines of failure.

(7) *Depressed Fracture with Evulsion*

After the line of fracture is established, bone and intracranial contents may become evulsed as a result of continuing movement of the striking object (*see* Fig 86*A*)

In accidents, multiple impacts are a possibility After the initial impact of the object on the head, or vice versa, the head may rebound, causing injury in an opposite area, such injury is not always due to a

contrecoup mechanism. Secondary injury to structures other than the head are also possible, for example, fracture of the first or second cervical vertebra caused by the downward push of the head from a blow to the interparietal area

Perforating and Penetrating Injuries

The bullets of military practice are usually jacketed with steel and have a core of lead, so that at velocities of 2,500 feet or more per second they still retain their shape upon impact with bone The bullets in civilian life are more often soft, and shatter upon impact The muzzle velocity of a rifle bullet may be 2,000 feet or more per second, the rifling causes the bullet to rotate about its long axis 3,000 times or more per second, giving it a velocity of about 500 feet per second The early theory that the explosive effect of a missile of high velocity was due to rotation about its axis, rather than to its velocity, has been disproved experimentally.[3]

Shell fragments, too, may have high velocities, estimated at 4,000 to 5,000 feet per second near the site of the explosion Large shell fragments may cause extensive laceration of the scalp and brain, while extremely small ones may be dispersed in the cranial cavity and cause relatively few clinical symptoms and signs. Contamination by clothing, hair, and helmet lining, and intracranial introduction of the contaminants is more likely to occur with shell fragments than with bullets

Experimental studies have clarified the various factors in the traumatic effect of high-velocity missiles Wilson,[74] for example, found that the wounding effect depended upon: (*1*) amount of energy transmitted to the tissues; (2) velocity of transmission; (*3*) direction of the energy; and (*4*) tissue density. The energy transmitted is calculated by the formula:

$$\frac{\textit{Mass} \times \textit{Velocity}^2}{2}$$

At velocities up to 2,000 feet or more per second, the energy, as evidenced by the damage caused, is proportional to the square of the

velocity The length of time it takes to transmit the energy to the tissues depends on the velocity of the missile, the shorter the time, the greater the energy per unit of time.

According to Callender,[7] however, at extremely high velocities the wounding effect varies as the velocity cubed and only as a single power of the mass. He, too, gave tissue density an important role in the resultant damage. The energy of a missile passing through a gelatin solution is dispersed inversely to the square of the percentage of density, for example, the explosive effects are four times as great in a 5 per cent solution as in a 10 per cent one, nine times as great as in a 15 per cent solution, and sixteen times as great as in a 20 per cent solution.[74] A high-velocity pellet without rotational acceleration entering a gelatin cube causes a tail splash at the entrance and marked swelling and enlargement of the cube (fourfold or more) as it passes through the cube, the enlargement is caused by a temporary cavitation, the cube returning to its original size after the pellet has passed through.[8] The rapid, temporary cavitation around the missile's path, also found in animal studies, is caused by the radial velocity imparted by the missile to the tissues through which it passes.[6] In the cranium with contents intact, the cavitation causes an intense, sudden increase in intracranial pressure, with tissue damage at a distance from the missile's tract, and extensive disorganization and fragmentation of bone and separation at suture lines. In the empty skull, the fragmented fracturing was absent, the perforation of entry being several times larger than the missile and that of exit only a little larger than the missile.[74]

In penetrating and perforating injuries due to high velocity bullets and shell fragments, the damage is caused by the intense increase in intracranial pressure, with shearing of tissue as the missile penetrates or perforates the skull.

In penetrating low velocity injuries, such as caused by knife blade, umbrella end, stick, or fan blade, there may be tearing of important vascular channels, a communication may be established between the cranial cavity and the nose, infection may be introduced into the cranial

cavity, but the brain about the tract of the injuring object is usually not extensively pulped and therefore it can successfully resist certain degrees and types of infections

Injury of Temporal Bone and Organs of Hearing and Equilibrium (Fig. 17)

The petrous portion of the temporal bone houses the middle ear and the membranous labyrinth. The facial nerve is intimately related to these structures, and passes through the temporal bone Injury of the temporal bone therefore frequently compromises all these structures. Linear fractures may extend from the temporal and parietal area into the mastoid process or into the external auditory canal and then bisect the petrous bone lengthwise A posteroparietal blow may injure either the mastoid process or the external auditory canal (*see* Fig 15) An anterior parietal blow may result in a fracture extending into the middle fossa, involving the greater wing of the sphenoid toward the fossa housing the gasserian ganglion, and the sphenoid sinus and pituitary areas Transverse fractures of the petrous bone usually result from occipital paramedian blows, occasionally, frontal blows may bisect this structure, with posterior extensions of the fracture from the anterior and middle fossae The facial and acoustic nerves are frequently involved in transverse fractures of the petrous bone, since the fracture courses through the internal acoustic meatus A discrete transverse or solitary longitudinal fracture of the petrous bone is uncommon, usually the fracture is a combination of transverse and longitudinal fracture because of the presence of many foramens and irregularities in the bone which constitute areas of stress concentration (Fig 17)

In one study, cadaver skulls were compressed from side to side in a vise.[31] The temporal bone was torn from the greater wing of the sphenoid and the petrous apex tilted medially and posteriorly The carotid canal and Meckel's cavity were demolished, with involvement of the superior petrosal sinus and the trigeminal and abducens nerves.

Compression of the head, anteroposteriorly, fractured the pyramid and the basilar process of the occipital bone, the fractures traversing the structures from the more forward portions or the more posterior regions of the skull, in an anteroposterior direction.

Aside from impact injuries, causing actual fracture lines to involve the petrous bone and its contents, indirect injuries from acceleration or deceleration also occur These may set up a pressure pulse in the membranous labyrinth, causing small hemorrhages and leading to vestibular and hearing dysfunctions. Such injuries may also be produced by boxing (*see* p 349)

With extensive transverse fracture and comminution of the temporal bone, or comminuted longitudinal fractures of the petrous bone, actual fragmentation and/or perforation of the vestibular and semicircular system and of the cochlea may occur

Various explanations have been offered for the deafness and vestibular disturbances which are frequent concomitants of head injury in which there is no roentgenographic or clinical evidence of skull fracture (*1*) hemorrhage into the labyrinth,[5, 23, 26, 32, 57, 61] (2) severe degenerative changes in Corti s organ,[12, 38, 43, 78] (*3*) cochlear nerve injury by stretching or tearing, due to stresses involving the petrous bone and to pressure waves in the cochleovestibular end organs, or in injuries involving the craniospinal junction.[24, 68, 69] Concussion may be responsible for loss of hearing,[2] it may not only cause hemorrhage into the inner ear[23, 26] but also injure the auditory pathways,[2, 53] the cochlear nuclei[53] or higher centers.

There is apparently no conclusive evidence that lesions of the central nervous system can cause true deafness,[18, 37, 43] despite suggestions to the contrary[64] Experimental bilateral destruction of the auditory cortex has no effect on auditory thresholds, and up to 75 per cent of the nerve fibers supplying a particular region of the cochlea can be destroyed without impairing the thresholds for frequencies having their locus in that region[39]

Moderate blows to the exposed mastoid cause mild dips in hearing

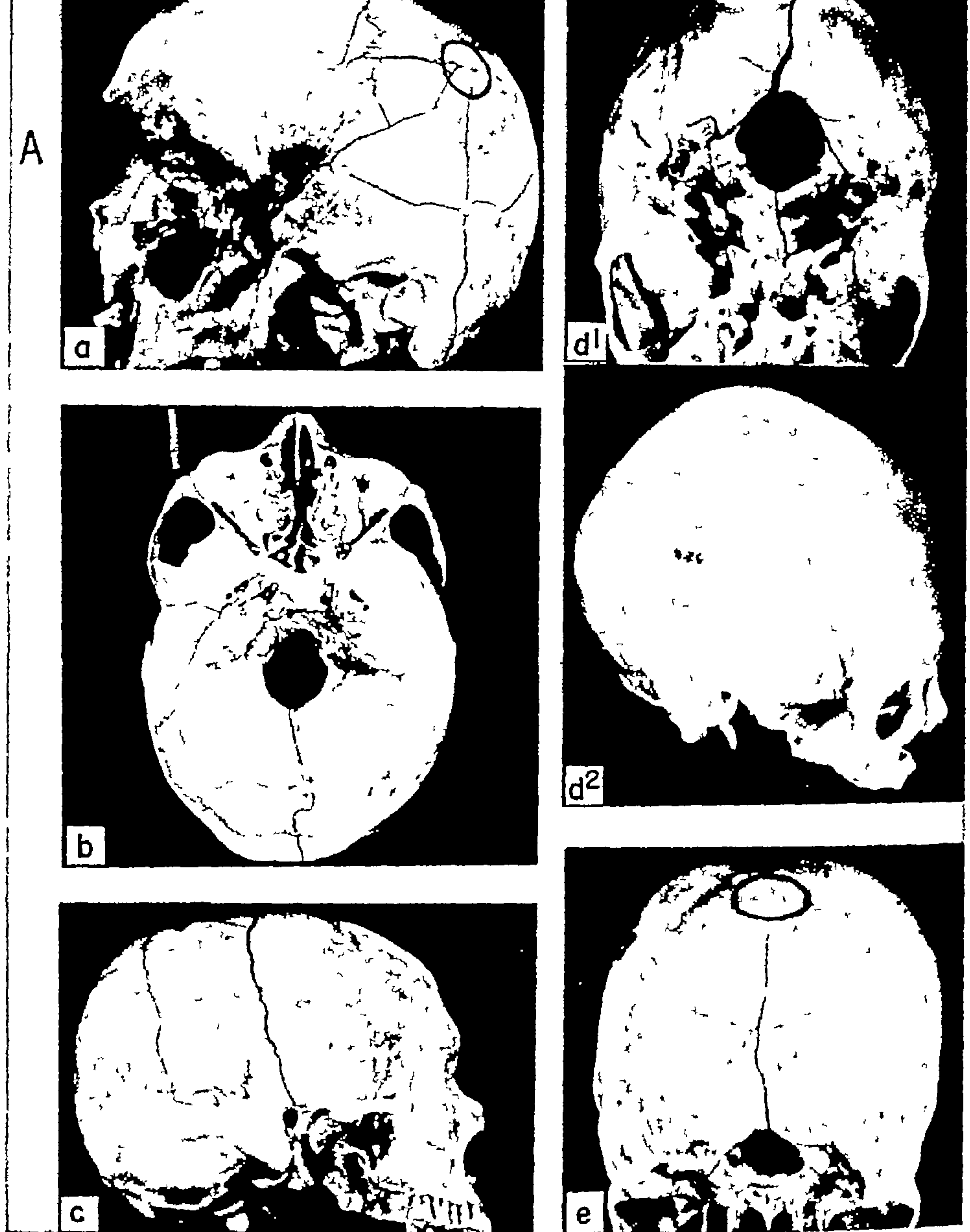


Fig 17. Petrous bone fractures (*A*) In cadaver skulls (*a*) Blow near vertex, with stellate fracture and extension to left mastoid (*b*) Blow to vertex, with midline occipital and left middle fossa fractures (*c*) Blow to vertex, with fracture extending into external auditory canal and parietal bone (*d*) Occipital blow, with midline occipital and basilar fracture, and transverse fracture of right petrous bone ending in anterior temporal area (*e*) Occipital blow, with occipital fracture and involvement of both condyloid processes (such extension may involve one or both petrous bones)

Fig. 17 (*continued*) (*B*) In patients (*a*) Parietal fracture extending perpendicularly into petrous bone and posterior fossa as triangular-shaped fracture of anterior medial border of foramen magnum (*b*) Perpendicular fracture extending toward and no doubt involving petrous bones lengthwise. (*c*) Depressed fracture of left frontal area extending across pituitary fossa, with tearing of optic nerve and carotid artery on left, and lengthwise into right petrous bone. (*d*) Longitudinal petrous bone fracture, found on operation for cerebrospinal fluid otorrhea, with tears of dura and superior petrosal sinus (fracture not visualized on roentgenograms).









B



acuity at 4,000 cycles for 24 to 48 hours,[14, 40] mallet blows at mastoidectomy may cause permanent hearing loss for this frequency[77] Experimental blows to skulls of cats cause deafness more frequently when the blow is near the ear, hearing acuity of 15 to 40 decibels is recovered during the first 2 weeks[58] There are no significant gross or microscopic lesions in the temporal bone; the primary injury is a degenerative change in Corti's organ, with secondary degenerative changes in the nerve fibers and ganglion cells supplying the cochlear traumatized area, with maximum nerve degeneration occurring 3 weeks after injury In these experiments, deafness was apparently not caused by hemorrhage into the labyrinth

Injury of Orbit and Visual Apparatus

A direct blow may tear or destroy tissues in the orbital area, the extent of injury varying from minor bruises to tissue loss Both direct and indirect injuries may be produced by acceleration or deceleration, as well as by compression of the head With deceleration, varying degrees of ecchymosis, orbital and periorbital hemorrhages, and even major ocular damage may occur

Blows to the forward portion of the vault can cause extensive deformation of the orbital roof on one or both sides (Fig 18) Stresscoat studies suggest that such blows set up tearing-apart forces from temple to temple. Comminution and depression of the orbital roof may occur, particularly if the bone is thin, and occasionally this may happen in closed head injuries The orbital floor may also be involved, the direction of the tearing-apart forces is then from side to side, as shown by the stresscoat experiments Fractures may thus extend through the infraorbital foramen, and involve the infraorbital nerve With blows of greater force, fracture and comminution of the orbital floor may occur.

Blows in the interparietal and lateral frontal regions create tearing-apart forces which cause fracture patterns extending toward the base (Fig. 18) and involving the middle fossa and the region of the pituitary

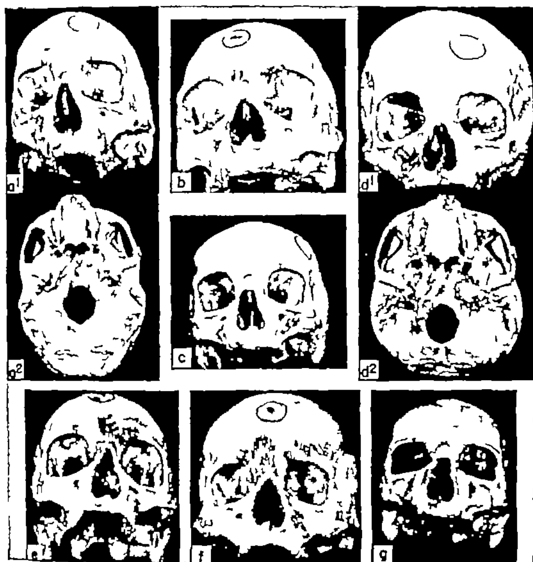


Fig 18. Stresses from frontal blows. (*a*) Blow to left of midline stresses in orbital roof, upper jaw and at base extending obliquely toward foramen magnum. (*b–c*) Stresses in orbital roof and upper jaw (*d*) Stresses in left orbital roof right upper jaw base around foramen magnum basilar process, and sphenoid body (*e–g*) Stresses mainly in one or both orbital roofs and upper jaw

fossa, and the sphenoid sinus. The fractures may also reach or involve the optic foramen. Blows in the more forward portions of the forehead may result in fractures involving the base of the anterior fossa and extending through the pituitary fossa into the basilar process of the occipital bone. Paranasal sinuses and the cavernous sinus may be involved, and the optic nerve may be hurt, the carotid artery may be

torn, resulting in an arteriovenous fistula in the cavernous sinus

In depressed fractures involving the medial orbital rim, the attachment of the superior oblique muscle to the trochlear fovea may be injured, resulting in abnormal movements of the eyeball

In head injuries by bullets, indirect orbital involvement may be due to the impact of the dural sac against the roof, particularly if the roof is thin, as a result of the intense increase in intracranial pressure at the time of penetration or perforation. In most cases, however, the tearing-apart forces in this area are in the opposite direction, so that the fractures are due to an upward rather than a downward rending of the roof It is noteworthy that President Lincoln's skull had an orbital roof fracture which was not associated with the area of impact or the path of the bullet; it may be recalled that the wound of entrance was in the right occipital area

Whiplash (Hyperextension, Hyperflexion) Injuries

Cranial and cervical spine injuries may occur as a result of rapid acceleration or deceleration of the body or the head in space The forward propulsion of the body from underneath the movable head causes hyperextension of the neck followed by flexion as the acceleration stops The whiplash injury has also been ascribed to an initial hyperflexion followed by extension.[20] This type of whiplash injury can occur when a car backs into the car behind it, or when a car is forcefully pushed into the car in front of it, in either case, the deceleration or arrest of the body is forceful If the head strikes a fixed object, such as the dashboard, a head injury is added to the neck injury.

The muscles at the craniospinal junction undergo severe strain, so that muscle fibers may be disrupted and small hemorrhages can occur The ligaments may be partly or completely evulsed, with stretching, tearing, and, possibly, hemorrhage The ligamentous capsules surrounding the facets, too, may be injured. The pulling, stretching, or com-

pressing of a nerve at the intervertebral foramen may result in nerve root injury The bony spinal canal and the intervertebral disk, together, may damage both the anterior and posterior roots One or more cervical nerves may be evulsed by a violent lateral movement of the head A predisposing factor in neural injury in this area is the presence of osteophytic disease about the foramens, vertebral bodies, and inter vertebral disks. The dentate attachments may also be a factor in the neural injury[33] Terms such as "traumatic cervical radiculitis" and "traumatic cervical neuritis have been used to describe the nerve in juries.

The cervical spinous processes may be fractured or evulsed,[21] but more commonly it is the bodies themselves which are compressed and/or dislocated, and occasionally torn or sheared off[56] Severe displacement of an intervertebral disk may compress the spinal cord[60] This type of major injury is more common in direct head blows, as in diving accidents, than in whiplash accidents. The brain seldom suf fers severe injury, being protected at the expense of the cervical spine, its muscles, and ligaments (*see* Fig *48B*)

Among the factors in the mechanism of whiplash injuries are (*1*) hyperextension or hyperflexion, with stretching of tissues, (2) tensile and compressive forces, with fracture of bone and tearing of tissues, and (*3*) acceleration or deceleration of the head, with intra cranial and temporal bone involvement. Other less common factors are (*4*) stretching of the carotid artery, with intimal injury and throm bosis, (*5*) stretching of the dural structures, including the junction of the spinal cord and brain stem, with severe brain stem involvement (*6*) cerebral mass movements due to acceleration or deceleration, with brain injury or brain stem involvement due to pressure gradients cre ated by a pressure build-up, (*7*) injury of the membranous labyrinth by the same forces, with involvement of the cochleovestibular mecha nisms, and (*8*) stretching of structures in juxtaposition to the spinal column with injury of the sympathetic chain causing pupillary inequalities and visual disturbances.

Cranial Nerve Injuries

Optic Nerve (Cranial II)

This nerve may be involved in closed head injury by: (*1*) contusion or transection at the site of a basilar fracture involving the optic foramen and/or the canal; (*2*) hemorrhages into the optic sheath; (*3*) compression due to deforming bony structures at the time of impact, resulting in nerve fiber injury, and (*4*) disruption of the nerve's blood supply, resulting in damage and dysfunction The optic canal extends for a distance of 4 to 10 mm. and contains the optic nerve and the ophthalmic artery The canal is 4 to 6 mm. wide, the diameter of the optic nerve is on the average 4 5 mm , consequently, the nerve fits snugly into the canal Deformation of bone around the canal, with or without fracture, may therefore injure the nerve by the suddenly applied forces Since the optic foramen, like other foramens at the base, is an area of stress concentration, a fracture line in the vicinity tends to extend toward this opening, involving the foramen and injuring the nerve

Actual evulsion of nerve may occur, so that the optic nerve is torn from the retina at the posterior end of the eyeball This may be due to a forward movement of the eyeball in certain deceleration injuries

Oculomotor, Trochlear, and Abducens Nerves (Cranial III, IV, VI)

The mechanism of injury to these nerves may be tension and stretching Uncal herniation, with dislocation of the brain stem, will stretch the nerves, particularly the sixth nerve, which has a long course along the base Cushing[12] stated that the abducens nerve may be involved from strangulation by the adjacent, transverse branches of the basilar artery which overlie the nerve The imprint of the compression of the nerve by the artery may be visible at postmortem study

The third cranial nerve is more frequently affected by uncal herniation, the involvement may be unilateral o [illegible] l, depending on the stage of the herni [illegible] whether or n [illegible] has been relieved.

The superior portion of the nerve, containing the pupillary fibers, is injured first, with progressive deformation, the nerve function may be completely impaired. Another mechanism of injury is compression of the nerve as it passes through the superior orbital fissure by an extra dural hematoma

Trigeminal Nerve (Cranial V)

The gasserian ganglion, the sensory and motor roots, or any of the divisions of this nerve may be injured Trauma of the gasserian ganglion is more common with blunt than with penetrating injuries.[31] Fractures of the anterior fossa may injure the nasociliary nerve.

Concussion Mechanism and Anatomic Pathology

Mechanism

Since the early eighteenth century, concussion has been defined as a state of unconsciousness following a blow to the head, with slow pulse and the pallor of a shocklike state but without visible lesions of the nervous system. (In the experimental animal, concussion is a posttraumatic unconscious state, judged by the apnea, vasomotor stimulation and loss of corneal reflexes following impact, *see* p 110) In our opinion, it is now well established that the brain stem, particularly the reticular formation, is involved in all cases of concussion, which may vary from a completely reversible state to one of continued coma ending in death The brain stem may suffer injury as a result of increased intracranial pressure at the time of impact, or be directly injured by distortion, mass movement of intracranial contents, shearing or the passage of a missile.

While the exact cause of unconsciousness after the impact of a blow to the head is still *not clearly understood*, *evidence is increasing that* the reticular formation of the brain stem, the periaqueductal gray matter, and the posterior parts of the thalamus and hypothalamus mediate an arousal or conscious state In the monkey electrocoagulation of

Cranial Nerve Injuries

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the cephalic end of the reticular formation causes a comatose state[78] Compression of the base of the brain stem during neurosurgery under local anesthesia frequently causes unconsciousness associated with deep snoring. The pacemaker mechanism for cerebral electric activity is located in the reticular formation of the brain stem, and its dysfunction causes disorganization of the rhythmic cerebral electric pattern It is believed that the unconsciousness of the anesthetic state derives from a depression of the cellular activity of this region by the anesthetic agents[1, 10] Traumatic involvement of this area resulting in cellular dysfunction may therefore be the cause of unconsciousness

The terms "concussion," "contusion," and "laceration" should not be used to denote varying degrees of central nervous system damage, with concussion representing the mildest form Concussion may or may not be accompanied by contusion or laceration If either of these occurs in the brain stem, the injury may be fatal, but contusions and lacerations elsewhere, even when serious, are not necessarily associated with concussion

In head injuries by a blunt object, which causes no major distortion of the skull and its contents, and in the absence of extensive bone failure, the effects of the blow can be ascribed primarily to a sudden increase in intracranial pressure resulting from the skull deformation and the acceleration or deceleration of the head's movements This pressure may be of the order of 750 to 5,000 mm. of mercury, or 15 to 100 lb per square inch, rather than the 300 mm. of mercury suggested by Scott[80]

In such circumstances, the brain stem, being near the craniospinal junction, suffers the greatest injury. The shearing stress occurring in this region is a function of the pressure produced and its duration (Fig 19). The anatomic characteristics of the head and the spinal canal to which it is attached (*see* pp 56-57) also play an important role: the head is a closed cavity solidly supported by bone in all directions except at the foramen magnum, whereas the structure of the spinal canal largely prevents the build-up of pressure in it.

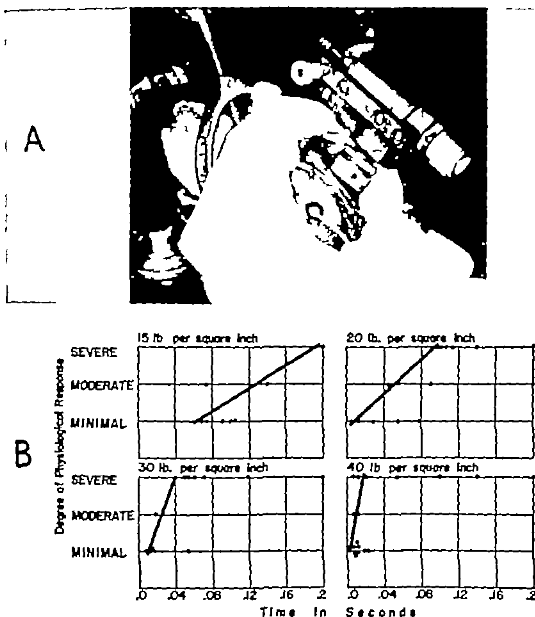


Fig 19 Effects of known air pressures applied at controlled times. (*A*) Technic. Device in place on anesthetized dog's head pressure pickup in left skull to record pressure for additional accuracy (*B*) Results. Each dot represents an experiment.

When a sudden increase in intracranial pressure occurs, a pressure gradient is created through the region of the brain stem and the cerebellum toward the foramen magnum and produces shearing stress. The effect of this pressure gradient differs from that produced by the

acceleration, which is in the direction of the acceleration; in the latter case, all elements in the brain are essentially in dynamic equilibrium, due to the action of inertial forces If this dynamic equilibrium were perfect, no shearing strains would be produced due to this pressure gradient However, since a blow deforms the skull, some shearing stress must occur throughout the cranial cavity, although of slighter degree than that at the craniospinal junction

The effects of acceleration were studied in anesthetized dogs by the Statham accelerometer which records the degree and duration of the acceleration, and the barium titanate accelerometer, which records the peak of the acceleration (Fig 20) The results obtained with the two instruments were essentially similar. The acceleration was produced by striking the head with a hammer. The intracranial pressure was measured by a pressure pickup screwed through threaded holes at various sites in the skull so as to make contact with the cerebrospinal fluid The accelerometer was so mounted on the head at a point opposite the area of the blow that the axis of the instrument was in line with the blow.

Acceleration was invariably accompanied by an increase in intracranial pressure However, there was no apparent correlation between the degree of accleration and the pathophysiologic effects, since some dogs manifested no concussive effects with high accelerations, whereas in others there were profound and even fatal changes with low accelerations Hammer blows not severe enough to cause depressed fractures caused high intracranial pressures at the site of impact and subnormal pressures in the contralateral area. With blows producing concussive effects, a correlation was found between the duration of acceleration and the increase in pressure, the seriousness of the effects increasing with the duration of the pressure.

The conclusion that the duration rather than the degree of acceleration causes the pathophysiologic effects therefore seems justifiable, particularly when the following experimental results are critically analyzed An air pulse of known magnitude was applied to the dural sac of the

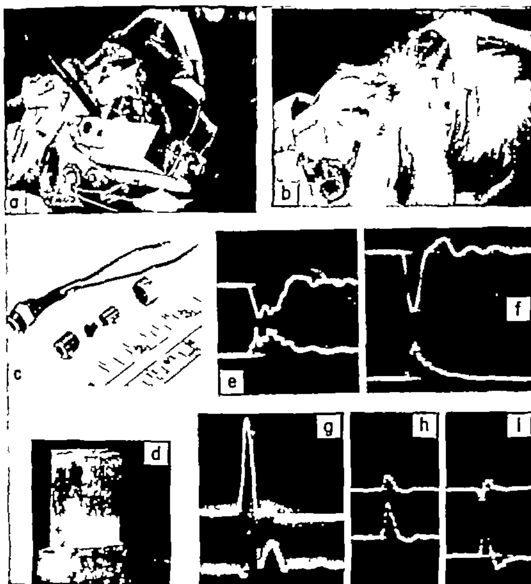


Fig 20 Acceleration and intracranial pressure. (a) Head of anesthetized dog with pressure pickup (c) and Statham accelerometer (d) in place. (b) Barium titanate accelerometer in place. (e–f) Acceleration (upper tracing) and pressure (lower tracing) (g) Midline blow: bilateral increase in pressure. (h) Blow near midline: higher pressure on side of impact than on contralateral side. (i) Blow on side: high pressure on ipsilateral side, subnormal pressure on contralateral side.

dog through an opening in the skull into which a piston-and-cylinder device had been screwed (Fig 19) As the piston was pushed through the cylinder by air at a fixed pressure, ports were alternately opened and closed, so that the external air came in contact with the dura Pressures

up to 100 lb per square inch could thus be applied to the brain for various periods, starting at less than 0 001 second It was found that pressure alone was not a significant factor in concussion, but that its duration was, for example, a low pressure (40 lb /sq in) for 0 1 second produced the same type of physiologic response as a high pressure (80 lb /sq in) for 0 01 second (Fig 19)

In summary, then, a sudden increase in intracranial pressure at the time of impact, particularly in blunt injuries, is caused by deformation of the skull, which reduces the cranial volume and increases the pressure within the cavity, and by acceleration or deceleration of the head, which also increases intracranial pressure, the increase being directly related to the degree of acceleration or deceleration

Anatomic Pathology (Fig. 21)

In our animals with experimental posttraumatic unconsciousness, we found an involvement of brain stem cells without similar involvement of cortical cells The most significant histologic change was central chromatolysis of the larger cells of the reticular formation of the brain stem The number of altered cells was greater in the medulla, whereas the cells in the pons and the midbrain were less involved. Lesions consisting of pontine hemorrhages and of swelling and fragmentation of axis-cylinders in the myelin sheaths were in most cases produced only by the upper extremes of pressure which were invariably fatal within minutes to hours The presence of cortical cell changes, such as described by Windle, Groat, and associates,[24 75] could not be established. since cells of similar appearance were found in the control animals Windle *et al*[24 75] had found chromatolysis of the cells of the reticular formation and a granular change in the large pyramidal cells of the motor cortex Our studies demonstrated that involvement of the upper brain stem increased with the increasing severity of the pathophysiologic effect

Abnormal alterations in the cell structure of the reticular formation were also seen in animals in whom the concussive effect was minimal

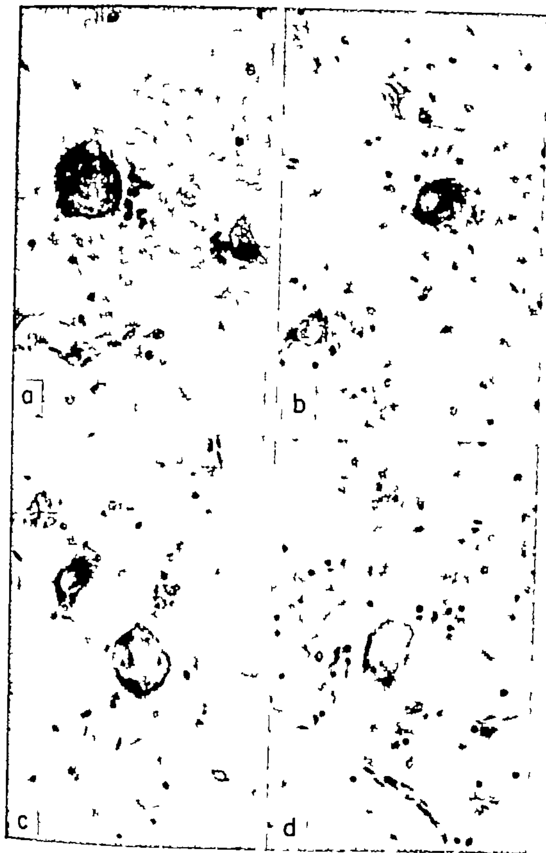


Fig 21 Cellular changes in experimental concussion, produced by sudden increases in intracranial pressure in dog's brain. (*a*) After subconcussive blow (*b*) After moderately concussive blow (*c–d*) After severely concussive blow

and in some who showed no change in blood pressure, respiration, and corneal reflexes, which are the criteria for a concussive effect Minor involvement of reticular formation cells suggests reversibility In man, the effects of a subconcussive blow may have an organic basis, improvement occurring as the cells recover from their damaged condition or as healthy cells take over the functions of the dying cells

The experimental study of concussion is more informative than evaluation of concussive changes in man, primarily because concussion alone is rarely fatal in man Furthermore, fixation of central nervous system tissue is inadequate for the accurate cytologic interpretations that are essential in this condition To this must be added the possible effects of preceding and accompanying diseases which alter the appearance of the various components of the central nervous system It is an extremely difficult, if not impossible, task to separate with assurance the histologic changes that are peculiar to concussion from other concomitant changes, including those due to other effects of trauma and those resulting from indirect and secondary cerebral involvement, such as caused by shock, hypoxia, fat embolism, and the toxic effects of infection in cases of fatal head injury

In summary, the posttraumatic unconsciousness of experimental concussion is always associated with alterations in cell structure in the brain stem, but without definite evidence of cortical cell involvement in either moderate or severe concussion.

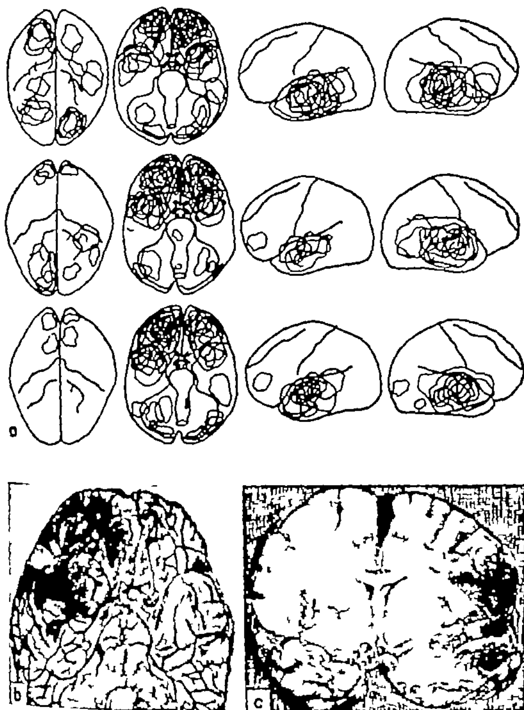


Fig. 22. Cerebral contusions. (*a*) Sites, as found in 151 cases of fatal head injury (*b*) Typical frontal and temporal contusions. (*c*) Deep contusion on lateral aspect of hemisphere.

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Cerebral Contusions and Lacerations

Contusions are common in head injury In the majority of cases, the bruises occur on the orbital surfaces of the frontal lobes, and at the junction of the frontal and temporal lobes of the hemispheres (Fig 22) As was noted by the older investigators[9, 30] and re-emphasized more recently,[10, 28, 35] the cause for a preponderance of contusions in these regions is the irregular surface of the orbital roof, the sharp edge of the lesser wing of the sphenoid, and the shelflike separation between the

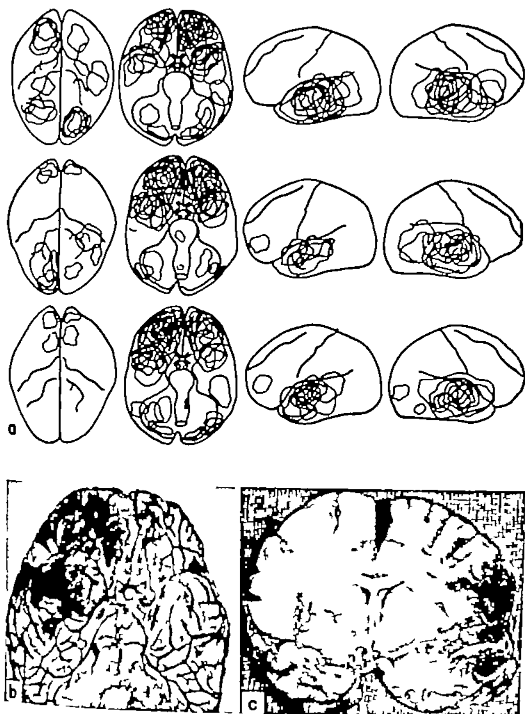


Fig. 22. Cerebral contusions. (*a*) Sites, as found in 151 cases of fatal head injury (*b*) Typical frontal and temporal contusions. (*c*) Deep contusion on lateral aspect of hemisphere.

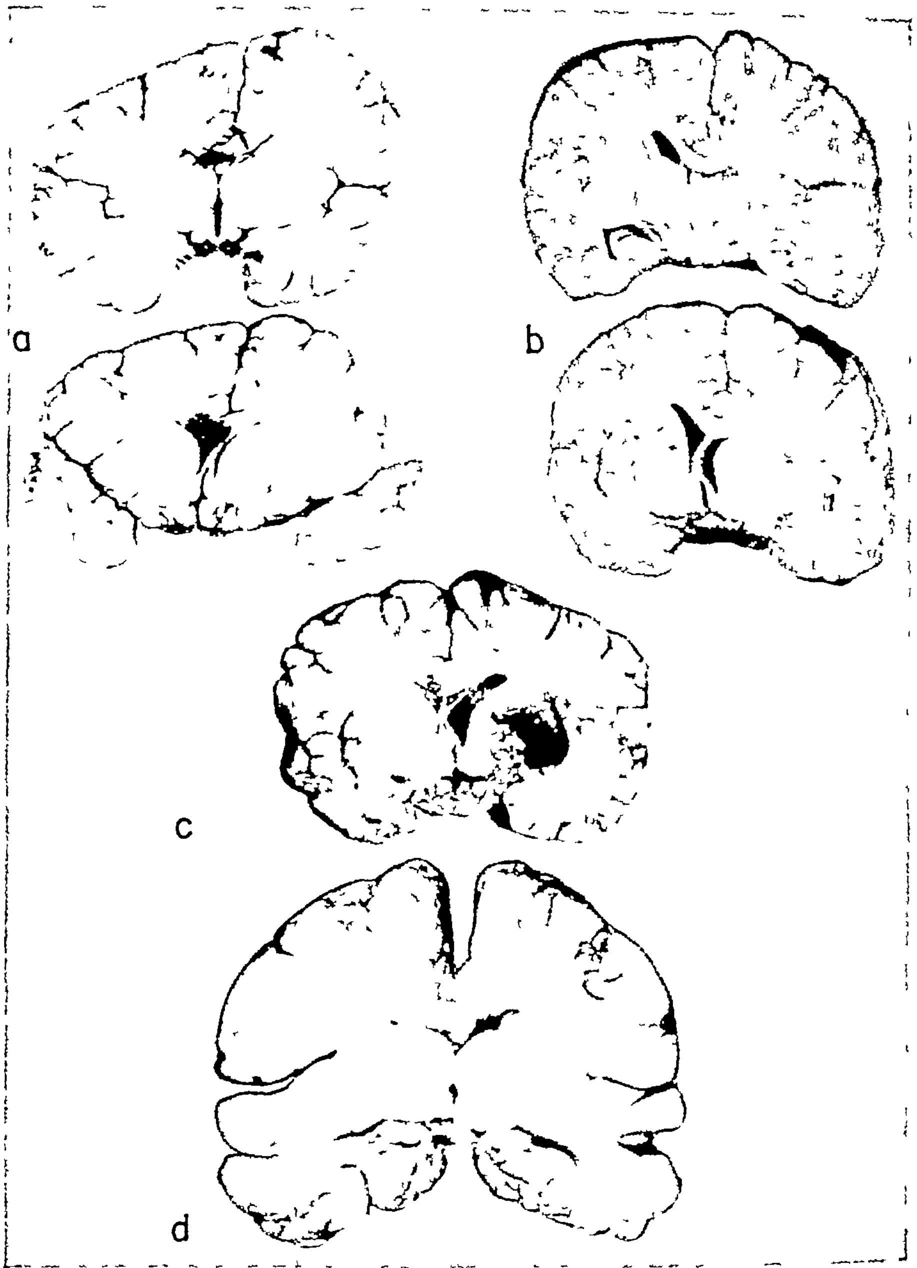


Fig 23 Cerebral lesions in head injury. (*a*) Severe contusion of corpus callosum. (*b*) Left-to-right migration of supracallosal gyrus under falx cerebri, thickened centrum ovale on left, and uncal herniation with involvement of both aspects of cerebral peduncle. (*c*) Contusion of corpus callosum and petechial hemorrhages in both hemispheres, note patent, larger ventricle on side of hematoma and compressed contralateral ventricle (*d*) Fat embolism, cross section showed petechiae throughout which proved to be fat globules in vessels.

anterior and the middle fossae, with the anterior fossa at a higher level than the middle one. Mass movements of intracranial contents cause the brain to strike these irregularities and thus be bruised Other causes of contusions and lacerations are (1) inbending of bone at the point of impact, with tearing and bruising of tissue at the site of fracture or by depressed bone fragments, (2) cavitation and subnormal pressures opposite the area of impact (contrecoup injuries), (3) skull distortion and cerebral tears resulting from deformation of the dural septums and the meningeal attachments to the brain, (4) shearing and tearing of attached areas when the brain is set in motion, with resultant disruption of connecting veins, and (5) shearing due to pressure gradients in the cranial cavity

Although bruises are common at the frontotemporal junction, other parts of the brain may also be involved, depending on the force and velocity of the injuring object. Certain structures which ordinarily serve for protection may be a source of injury The falx cerebelli supports the occipital lobe of the hemisphere and the superior surface of the cerebellar lobes. In severe injuries, its free edge or the incisura may tear or contuse the surface of the brain stem, causing irreparable damage. The inferior surface of the occipital lobe and the superior surface of the cerebellar lobes may be bruised or torn. The corpus callosum may be injured by the free edge of the falx cerebri. In other instances, mass movements of the brain in a downward direction near the midline may cause compression of the corpus callosum by the overlying cortex, with consequent contusion (Fig 23)

Many contusions are associated with small petechiae surrounding the main area of contusion (Fig 24) The microscopic features of a contusion are complete destruction of cells, capillary and vessel wall injury, and extravasations of blood into the tissues. Depending on the depth of the bruise, and the trauma of the vascular walls, an extensive area of softening necrosis, and infarction may occur and lead to varying amounts of further bleeding This large area of damage may be mistaken for an intracerebral hematoma.

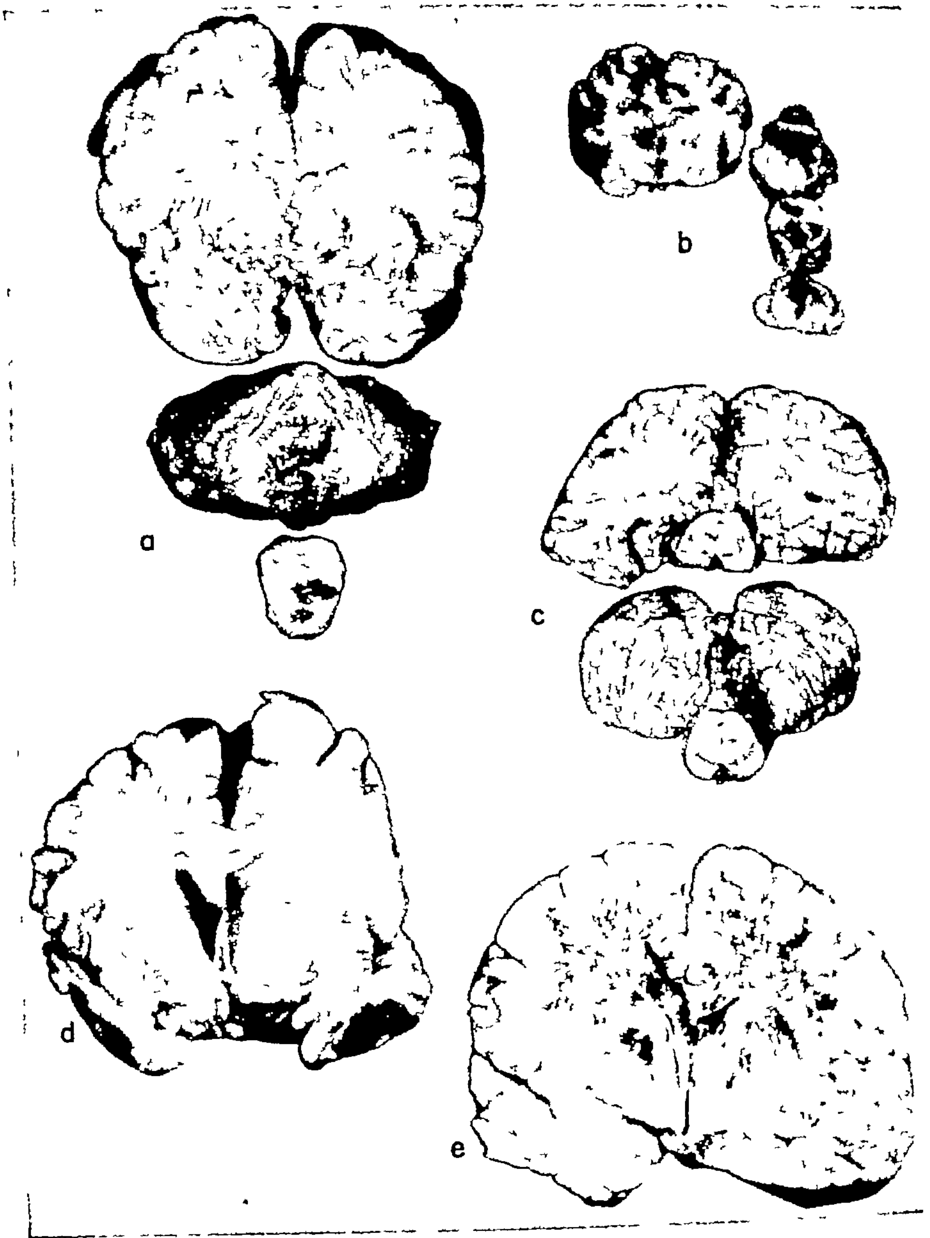


Fig 24 Petechial hemorrhages in head injury (*a*) Petechiae in posterior thalamus, midbrain, and upper pons, patient dead soon after injury (*b*) Similar petechiae in experimental concussion in dog (*c*) Petechiae in midbrain, contusion of right occipital lobe (*d*) Petechiae around contusion in left temporal lobe (*e*) Petechiae of fat embolism, note involvement of white matter, sparing of gray

Contrecoup Injuries

The cerebral surface may suffer a contrecoup injury from mass movement of intracranial contents or from cavitation in an area opposite to that of the impact[22] (Fig 22) As we have shown experimentally, an increase in intracranial pressure in the area of impact may be accompanied by a lowered pressure on the opposite side. When below normal pressures occur, vapor bubbles, resulting from the lowered pressures, break or collapse cavitation then occurs, causing small bruises and hemorrhages.

Blows to the frontal or occipital regions cause contusions of the frontal lobes on their inferior surfaces or of the temporal lobes with equal frequency Brain and bone are in intimate apposition in the frontal areas and mass movements due to frontal blows crowd the frontal and temporal lobes against the forward parts of the skull Subfrontal and temporal lobe contusions associated with occipital blows may be caused by the subnormal pressures, with resultant cavitation in the forward portion of the cranial cavity or by the forceful contact between the cranial contents and the irregularly formed anterior and middle fossae as the contents rebound from the impact The occipital and posterior portions of the brain on the other hand being encased in smooth bone, dura, and the tentorium cerebelli which has a cushioning effect, suffer fewer contusions from mass movement of the intracranial contents. From the evidence at hand, therefore, it would appear that both cavitation and impingement of the brain against irregular bony surfaces can cause contrecoup injury

Increased Intracranial Pressure and Cerebral Edema

The increase in pressure which occurs *after* the initial injury should not be mistaken for the sudden intense increase immediately upon impact. The increased pressure under discussion here may result from

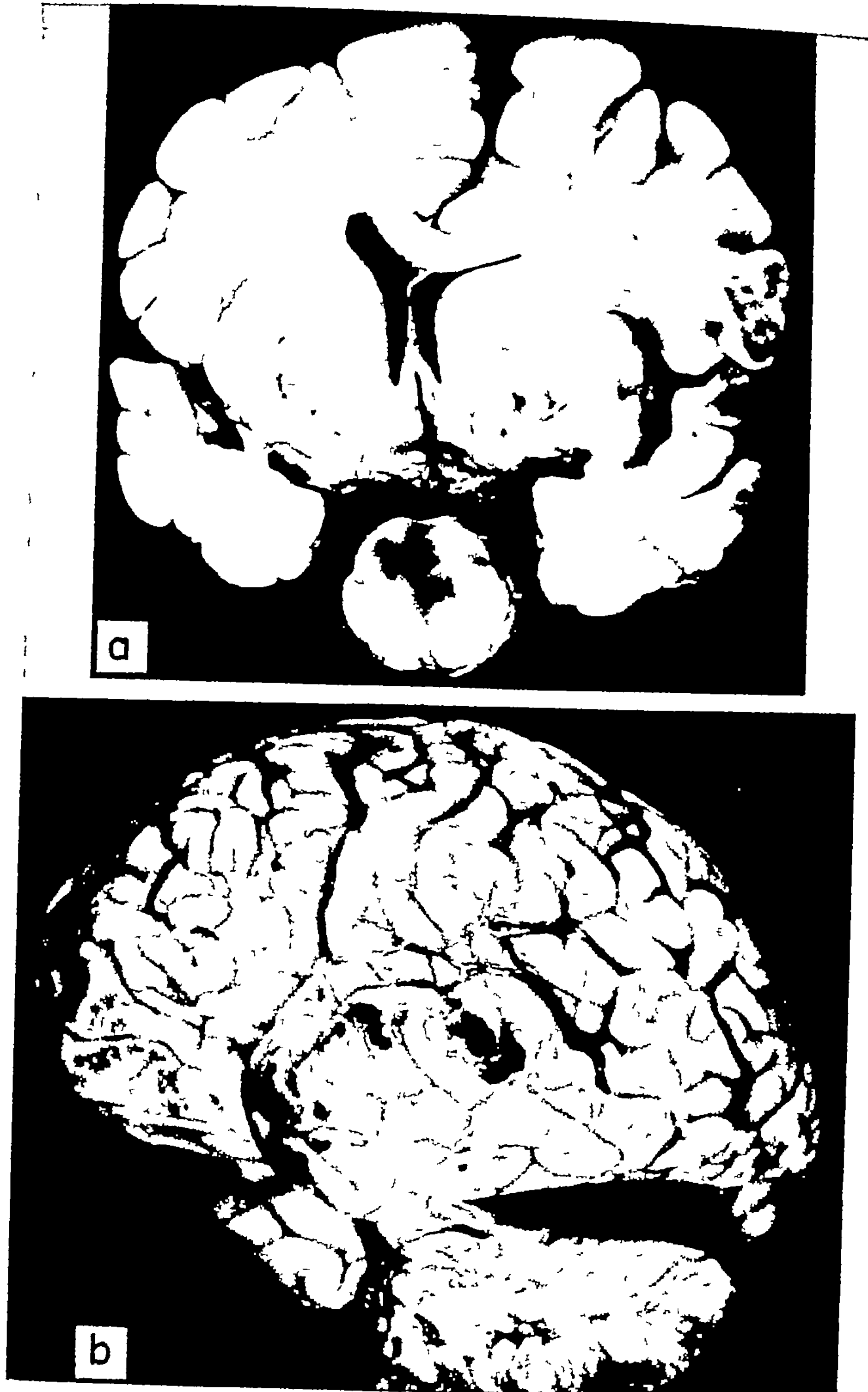
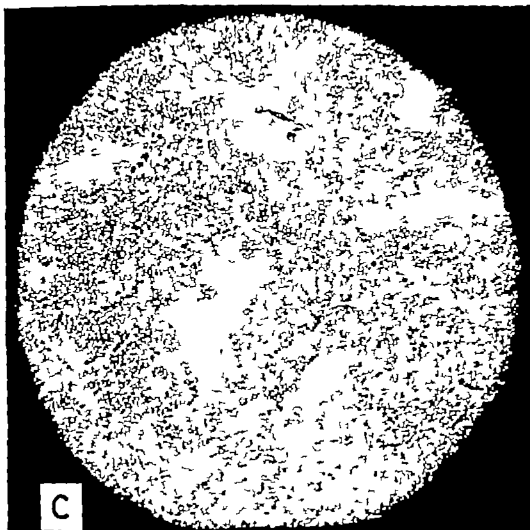


Fig 25 Cerebral edema (*a*) Left hemisphere thicker than right, marked shift of ventricular system and left-to-right shift of paracallosal gyrus, left cerebral surface contused, cause of death, hippocampal and tentorial herniation, with hemorrhages in upper brain stem (*b*) Cerebral contusions which can cause cerebral edema (*c*) Microscopic appearance of cerebral edema, note increased tissue spaces and intercellular fluid collection, × 90, hematoxylin–eosin stain



cerebral edema, or from hemorrhage into the intracranial contents with a resultant increase in intracranial blood volume, particularly in the subarachnoid space. The osmotic pressure of blood in the subdural and subarachnoid spaces or on the cerebral surface is high, this facilitates the collection of tissue and cerebrospinal fluids. Cerebral contusions, extravasation of blood, small thrombi and necrosis all may cause traumatic edema in the injured area, similar to that elsewhere in the body Migration of one or another part of the brain or uncal herniation as a result of swelling is common, as is thickening of the white matter (Figs. 23, 25) Browder and Rabiner[4] have described such an edema in the subcortical white matter directly beneath a mass lesion.

Edema in the absence of appreciable bleeding into the tissues is difficult to explain, however Some 60 years ago, vasomotor paralysis of cerebral vessels by trauma, with subsequent venostasis and waterlogging of tissues due to an increased permeability of the capillaries, was proposed as a cause of cerebral swelling[11] This mechanism has been held to be important in severe head injury by a number of investigators[15, 48, 55] An area of localized edema, by compressing the brain, alters its nutrition, so that the edema may be enhanced by anoxemia and tissue anoxia, leading to a generalized edema[54] Probably, several factors combine to cause a generalized cerebral edema in the absence of extensive intracranial hemorrhage increased osmotic pressure of the cerebral tissue and increased cerebrospinal fluid pressure, together with an inadequate oxygen supply A number of explanations have been offered for the occurrence of increased intracranial pressure when there is no hemorrhage (*1*) irritation and stimulation of the choroid plexus, resulting in increased formation of cerebrospinal fluid,[46] (*2*) lowered rate of cerebrospinal fluid absorption due to venostasis, with venous pressure increased well above that of cerebrospinal fluid, (*3*) increase in the protein content of the cerebrospinal fluid due to blood, retarding its absorption[71] Both Haussler[29] and Jorns[32] differentiated between cerebral swelling and edema Some workers found no increase in the water content of traumatized brains in animals The increase in the brain's intravascular and intraventricular fluid contents contributes to cerebral swelling[44, 61]

Pathophysiologic, Electric, and Chemical Changes

Pathophysiologic Changes (Fig. 26)

In the dog, a blow with minimal concussive effect (200 to 250 inch pounds of energy) causes a short period of apnea or of increased respiration, a rise or fall in blood pressure, and a brief loss of corneal and pupillary reflexes With increasingly strong blows, the duration and severity of the reactions increase Whereas respiration usually reappears

within 60 seconds in moderate concussion, the respiratory arrest is long or permanent in *severe concussion, the corneal reflexes do not return*, and the blood pressure rises steeply, drops after 2 or 3 minutes, rises

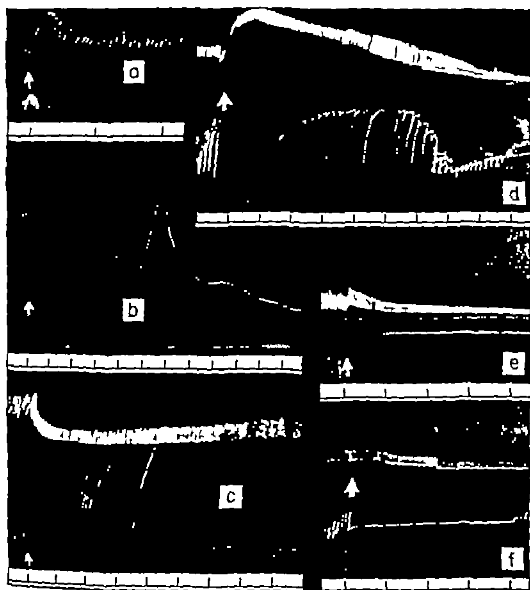


Fig 26. Pathophysiologic effects of experimental head injury (*A*) Increased respiration and rise in blood pressure in minimal concussion (*a*) Arrested respiration and steep rise in blood pressure in fatal concussion (*b*) Drop in blood pressure (*c*) frequently seen in depressed fractures with communition of bone and torn dura. *Brisk rise in blood pressure in fatal concussion in adrenalectomized* dog (*d*) No rise in blood pressure with fatal blow in yohimbinized dog (*e*) No rise in blood pressure after fatal blow to dog with cervical cord section (*f*) (*Continued on next page*)

Physiological Effect

PROFOUND

MODERATE

MINIMAL

MECHANICAL DRILL

AIR GUN (non-penetrating)

22 BB REVOLVER 780 ft/sec.

22 SHORT RIFLE 970 ft/sec.

a

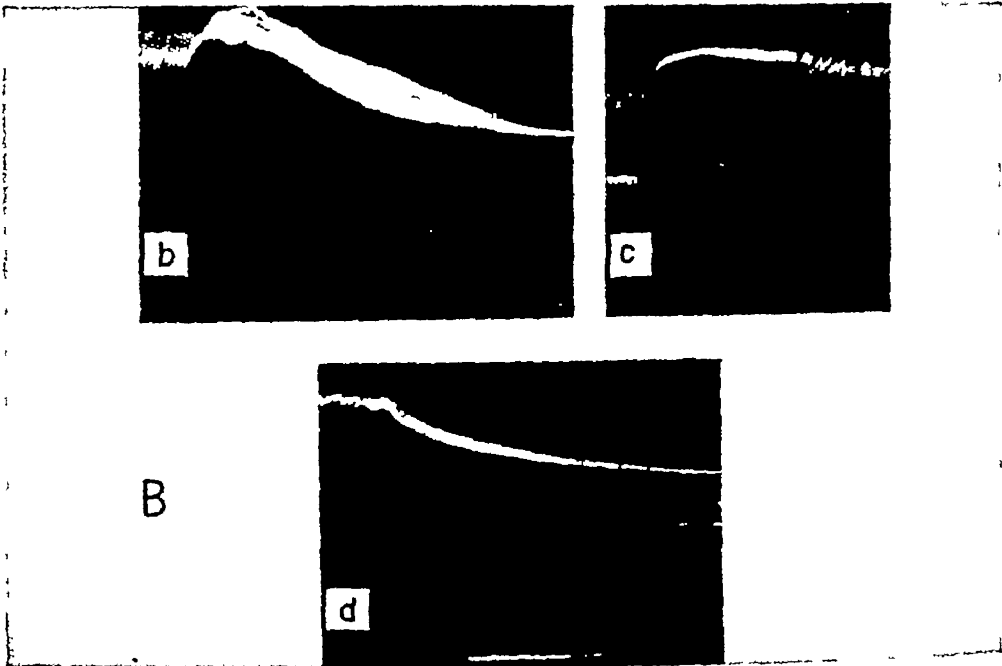


Fig. 26 (*continued*) (*B*) Extent of injury in relation to injuring object mechanical drill, air gun, or size and velocity of bullet (*a*). Initial increase in blood pressure and drop to zero, and stoppage of respiration in profound injury by gunshot (*b*). Increase in blood pressure for 3 to 4 minutes and short period of apnea in moderate injury by bullet (*c*). Drop in blood pressure without initial rise and sustained apnea in bullet injury of the medulla oblongata (*d*).

again, and then drops to zero The pulse rate is usually increased, but a slow rate (vagal effect), with escape, has also been noted frequently There is no extensor rigidity under general anesthesia, such as occurs after a blow to an animal under morphine analgesia.

The same pattern of response is obtained in decorticate and decerebrate (by section of the upper brain stem) animals The evidence that the rise in blood pressure in concussion is caused by peripheral vasoconstriction is supported by results obtained with various blows to the heads of adrenalectomized and yohimbinized dogs In serious injury, the peripheral vasoconstriction is associated with dilatation of the intracranial blood vessels,[18, 45] this difference in vascular tone insures an adequate blood supply to the brain stem Or the dilatation may be caused by the steep rise in blood pressure which distends the intracranial vessels

The peripheral vasoconstriction which occurs immediately after a moderate to severe injury may have significant clinical effects on the body economy anemia of various organs, particularly the kidneys and the gastrointestinal tract, with nephrosis, and gastrointestinal ulceration. From the available evidence, one may conclude that death soon after severe head injury is caused mainly by paralysis of the vasoconstrictor and respiratory centers due to brain stem injury (Fig 27), and that it is the paralysis of the former rather than stimulation of vasoglossopharyngeal centers which causes the failure in blood pressure.

Electric Changes

A blow to the head may injure the semipermeable membrane of the brain cells and briefly disturb the intracellular metabolism This may be accompanied by an increase in electric activity, to be followed by a period of depressed activity[71] The increased activity, it has been suggested is an injury potential.[41] After a concussive blow, the electric activity of the brain stem has been reported to be definitely slowed and that of the cortex impaired The reactivity of the brain stem to external stimuli, such as sciatic stimulation, is also impaired after a blow[17]

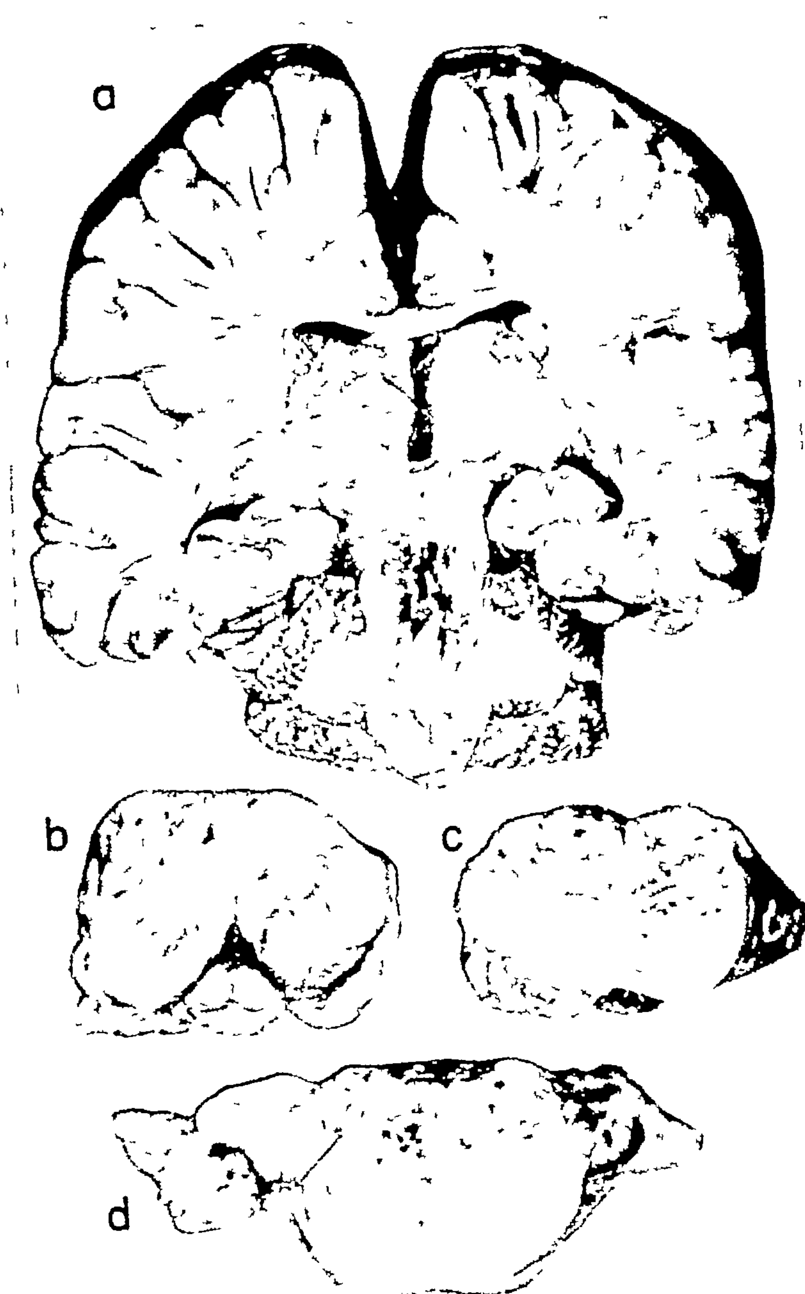


Fig 27. Fatal brain stem injury in 40 year old man, dead on arrival at the hospital Coronal and cross sections revealed petechial hemorrhages in upper brain stem.

Experimental concussion lowers the convulsive reactivity of the brain and impairs the electric activity of the supranuclear motor system,[illegible] metrazol induced convulsions in rats stop and sufficient atropine to block the acetylcholine effect on electric activity does not affect the interruption of convulsions.[62, 63] The electric activity is also affected by experimental, localized pressure on the dura but this may be due to pressure on the brain stem influencing the cortical discharges[22]

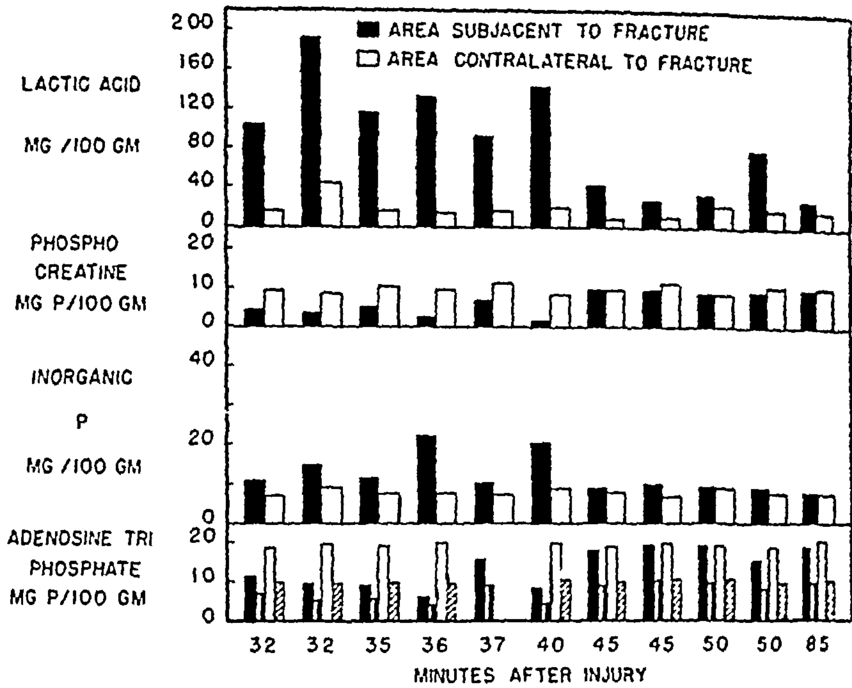
Chemical Changes

Head injury alters the concentrations of various substances in cerebral tissue (Fig 28) In experimental contusion, the *lactic acid* level rises markedly above the normal 15 mg per 100 cc 30 to 80 minutes after injury, and remains at a high level until tissue repair sets in and oxygen uptake by the contused tissue increases the return to normal levels occurs within a few days. The *phosphocreatine* and *adenosine triphosphate* levels decrease (normal, 15 and 18 mg / 100 cc., respectively), while the *inorganic phosphate* level rises all return to normal as tissue repair occurs

In experimental concussion, the concentrations of all three substances are normal, indicating that concussion does not materially affect the oxygen supply or uptake.[27] Increased oxygen consumption has been reported[28] but there is good reason for assuming that anoxia does not play a role in the concussion syndrome.

Acetylcholine and certain enzymes able to split nucleic acid and produce chromatolytic changes in the anterior horn cells *in vitro* have been reported to be present in the cerebrospinal fluid in experimental head injury[34] and in patients with head injuries.[illegible, 72] Other investigators found only minimal levels or none at all.[67] Disturbances of intracellular metabolism by the injury might account for these findings.

Glutamic oxaloacetic transaminase it has been reported, appears in the cerebrospinal fluid after cerebral infarction, head injury, and other cerebral lesions much more rapidly than in the blood[70]



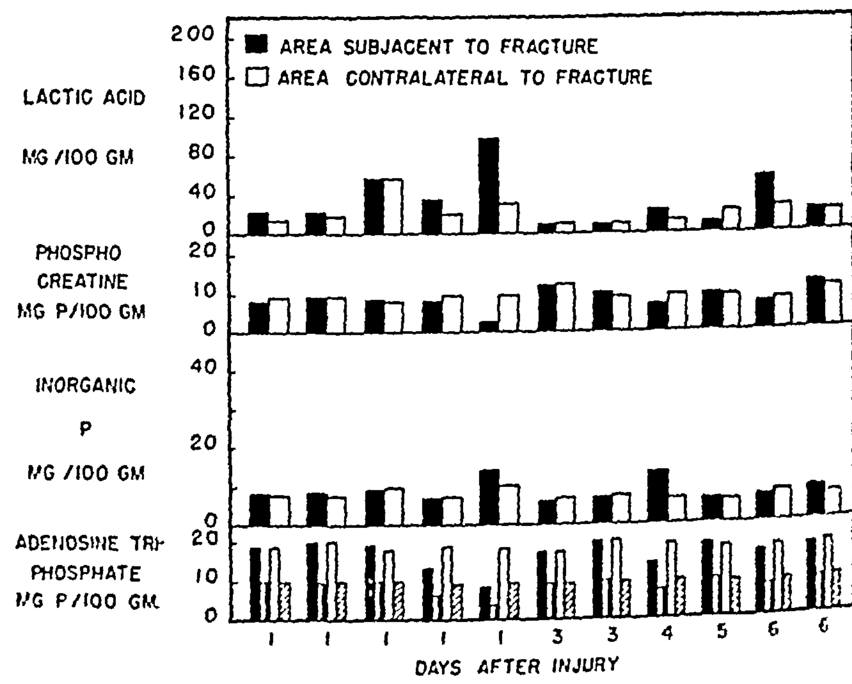


Fig 28. Chemical effects of experimental head injury. (*A*) Immediately after acute injury (*B*) One to six days after injury.

Injuries of certain parts of the brain may also cause blood chemistry changes, for example, higher levels of sodium and potassium with a parapituitary lesion impairing elaboration of the antidiuretic hormone, or, involvement of the preoptic area in a hypothalamic lesion, with osmoreceptor changes (*see* p 203)

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Chapter IV

DIAGNOSTIC TECHNICS

Roentgenography

Roentgenography is essential in the evaluation of almost every type of head injury and should be performed if at all possible in every case at the earliest opportunity. In some cases, however, delay is advisable. In large hospitals where the admitting and radiology departments are in close proximity, roentgenography can be carried out at the time of admission. Survey roentgenograms of the skull are invaluable; if the patient's condition permits and indications exist, roentgenography to locate other sites of injury can be performed at the same time. In high velocity injuries, or in injuries suffered in falls from a height, roentgenograms of the complete spine and the pelvis are important.

Routine roentgenograms of the skull should include the antero-posterior, posteroanterior, right lateral, and left lateral views. Special views, such as Stenver's, Mayer's, Schuller's, and Chamberlin's, may be of value in visualizing fracture of the temporal bone. Stenver's view is the projection of choice for visualization of a transverse fracture of the temporal bone. Some advise routine views of the orbital foramens. Roentgenograms of the base and stereoscopic studies may be indicated

in some cases. Magnification roentgen technics are helpful occasionally.[20] It is often advisable to call the radiologist's attention to the area of suspected fracture, so that special views may be obtained. Although visualization of fractures of the petrous portion of the temporal bone is often difficult, with care such fractures can sometimes be revealed. In patients with serious injuries, respiratory obstruction during the examination must be avoided, particularly when placing the patient in the prone position.

Roentgenography is of major importance in delineating the type of depression in cases of depressed fracture, and in planning the operative procedure. In penetrating head injuries due to high-velocity missiles, roentgenography reveals, in addition to fracture and comminution of bone, the presence of foreign matter and bone fragments in cerebral tissue. Even when there is only an apparently minor scalp wound or a laceration of the back of the head and neck, the roentgenograms may show extensive comminution and depression in the occipital area. After surgical treatment of such wounds, the postoperative roentgenographic studies help to determine whether the debridement was thorough enough.

The roentgenographic features of an *extradural hematoma* of middle meningeal artery origin are: (*1*) almost invariably a linear fracture, usually crossing the middle meningeal groove(s); (2) depressed bone in the parietotemporal region in some cases, and (*3*) shift of the pineal gland, an important feature since it may indicate the side of the lesion. The absence of a fracture line does not exclude the possibility of extradural hematoma, which may occur without skull fracture or with one so slight as not to be visible on the roentgenogram.

The features of *subdural hematoma* are: (*1*) a shift of the pineal gland; (2) occasionally, evidence of a linear skull fracture; (*3*) erosion or thinning of bone in some cases; (*4*) an internal calcific lining, if the hematoma has become calcified; (5) possibly, asymmetrically developed anterior skull if the hematoma is of the relapsing, juvenile form.

If a whipl[illegible] [illegible]y is [illegible] routine survey roentgenograms

may help to establish the diagnosis. Absence of a fracture or dislocation in this region usually is an indication that the injury is not severe. Occasionally, the roentgenograms disclose fracture of a body, facet, spinous process, lamina, or odontoid process. The roentgenography must be meticulous, all 7 cervical bodies and the odontoid process must be visualized, and views in flexion, extension, and in the erect position are helpful, uncovering slight subluxations which are not well visualized in conventional views. Narrowing of an intervertebral disk space may indicate a herniation of the nucleus pulposus at that point, but this is not a dependable sign.

The presence or the suspicion of herniation of cervical nucleus pulposus delays insurance settlement. A definite diagnosis is therefore important, and can be established with the help of myelography The Pantopaque must be removed after myelography, and during the procedure care must be taken to prevent entrance of the substance into the cranial cavity

Angiography

Since the introduction of angiography by Egas Moniz in 1927, it has become an important aid in the diagnosis and management of intracranial mass lesions.[12, 20, 22] If there is reason to suspect that a mass lesion may be present when the initial skull roentgenography is being done, the angiography can be carried out immediately thereafter, and the patient is thus spared a second trip to the radiology department. Angiography may be more effective than diagnostic trephination in establishing definitely the presence of a collection of blood or cerebrospinal fluid[24] While this technic, like other diagnostic methods, entails a certain risk, there are few contraindications to its use. The radiopaque substances used for cerebral angiography are Thorotrast, Diodrast, and, most recently, Hypaque.[20, 24]

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Technic

The contrast medium is injected into the internal carotid or common carotid artery, preferably bilaterally, or into the vertebral artery in some cases The older method of injecting the medium was by the open technic, in which the artery was exposed surgically The newer method, now used almost exclusively, is the closed, or percutaneous technic For this method, Xylocaine hydrochloride (lidocaine hydrochloride) is infiltrated locally, and Pentothal sodium (thiopental sodium) or Nembutal sodium (pentobarbital sodium) intravenously for anesthesia The contrast medium, 8 to 10 cc for each exposure, is then injected, and anteroposterior and lateral angiograms, with 0 125 second exposure, are taken, in some cases, serial Fairchild studies are also helpful Better visualization is sometimes obtained by using 15 to 20 cc of contrast medium

For injection of the vertebral artery, an 18 gage Touhy needle is inserted into the artery, usually at the fifth cervical interspace level

Findings (Fig. 29)

In the presence of *extradural hematoma,* the vessels on the surface of the convexity may be compressed, if the hematoma is at the base, the middle cerebral artery is pushed medially and upward, and the anterior cerebral artery is shifted toward the opposite side A *subdural hematoma,* too, may compress the vessels of the convexity, and cause a shift of the anterior cerebral artery toward the opposite side A frontal extradural or subdural hematoma causes the anterior cerebral artery to shift medially A frontal *intracerebral hematoma* widens the space between the anterior and middle cerebral vessels in the anteroposterior view Parietal and frontal hematomas depress the middle cerebral blood vessels The characteristic pattern of temporal lobe hematoma is a bowing and elevation of the middle cerebral blood vessels around the mass, in the anteroposterior views the inferior portion of the anterior cerebral artery may be shifted to the opposite side

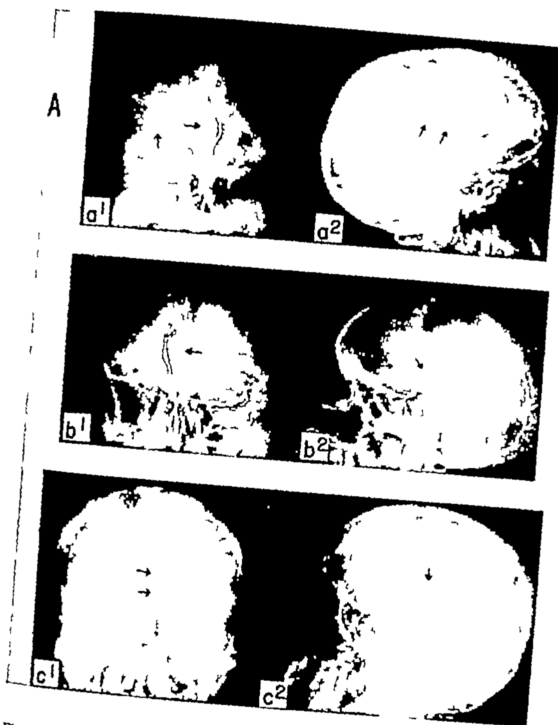


Fig 29 Angiography in head injury (*A*) Temporal extradural hematoma (*a*) anteroposterior view (a^1) upward and medial shift of middle cerebral artery and its branches, minimal shift of middle third of anterior cerebral artery lateral view (a^2) compression upward of middle cerebral group of vessels, arching over hematoma. (*b*) Left frontal lobe intracerebral hematoma anteroposterior view (b^1) widened space between middle and anterior cerebral arteries lateral view (b^2) depression of middle cerebral vessels. (*c*) Subdural hematoma anteroposterior view (c^1) left shift of anterior cerebral artery lateral view (c^2) downward shift of middle cerebral artery (*Continued on next page*)



Fig 29 (*continued*). (*B*) Frontal lobe intracerebral hematoma (*a*), anteroposterior view (a^1), shift of anterior cerebral artery across midline and increased space between anterior and middle cerebral arteries, lateral view (a^2), downward shift of middle cerebral vessels (*b*) Parietal extradural hematoma of middle meningeal origin; anteroposterior view (b^1), marked compression of cortical vessels, lateral view (b^2), slight compression of middle cerebral vessels (*c*) Left temporal lobe intracerebral hematoma simulating extradural hematoma, anteroposterior view (c^1), anterior cerebral artery shifted somewhat across midline, lateral view (c^2), marked upward shift of middle cerebral vessels.

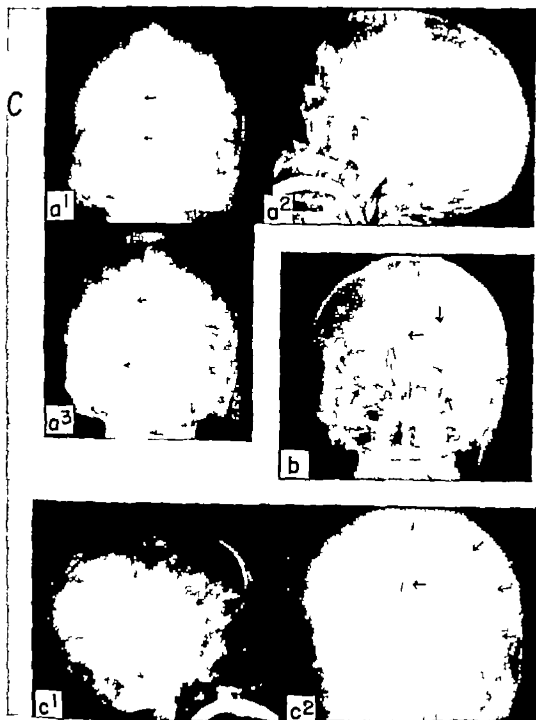


Fig. 29 (*continued*) (*C*) Acute subdural hematoma (*a*) note space between skull and blood vessels. (*b*) Chronic left subdural hematoma. (*c*) Subacute subdural hematoma note right shift of anterior cerebral artery and compressed vessels on left over hemispheral surface, at a distance from overlying bone. (*Continued on next page*)

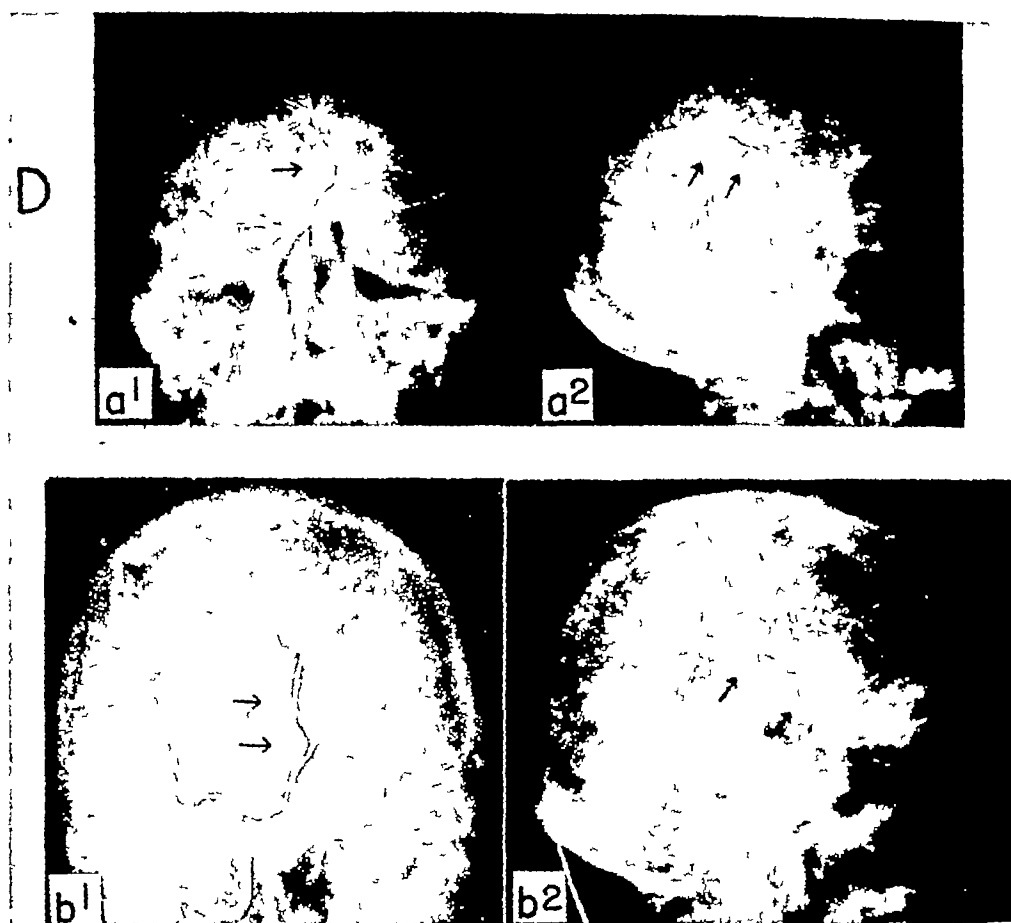


Fig 29 (*continued*) (*D*) Temporal hematoma (*a*), note left shift of anterior cerebral artery (a^1), and upward shift of middle cerebral vessels in lateral view (a^2). (*b*) False positive angiograms in patient suspected of having right temporal lobe intracerebral hematoma, autopsy revealed contusion, and edema of temporal lobe

Carotid artery angiography demonstrates a peripheral or intracerebral mass in the frontal, parietal, or temporal regions It is diagnostic in a high percentage of cases of chronic subdural hematoma.[23] A mass in the posterior parieto-occipital areas may be missed, and lesions in the posterior fossa are not visualized by a carotid angiogram; these lesions are not common, but they can be diagnosed by combining angiography with ventriculography by the parieto-occipital route

A normal angiogram in a patient with localizing signs is a strong indication of the presence of a cerebral contusion rather than of a mass

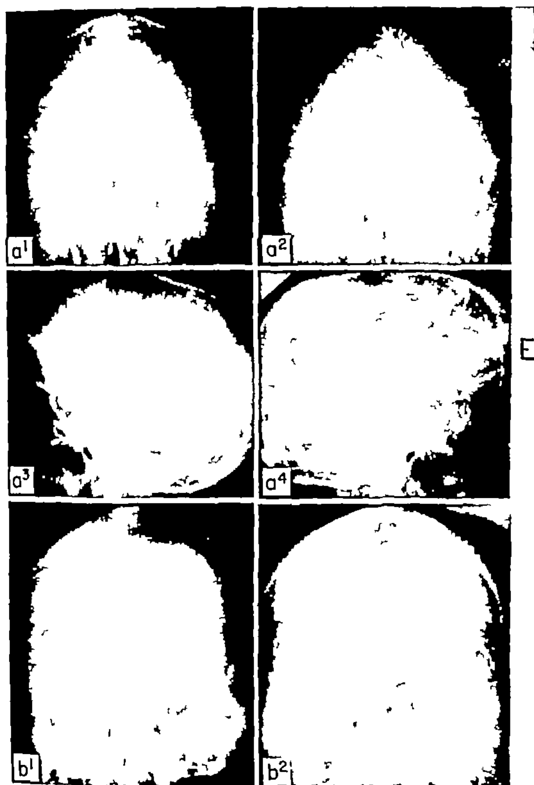


Fig 29 (*continued*) (*E*) Bilateral chronic subdural hematoma (*a*) note shift of anterior cerebral artery to right (a^4) (*b*) Subdural hematoma and intracerebral hematoma in left temporal area note shift of middle cerebral artery (b^2), with space between bone and brain due to subdural collection.

lesion Occasionally, a false positive angiogram is due to a localized hemispheral swelling While this may lead to an unnecessary operation, angiography compares favorably with diagnostic trephination, in which there is a higher percentage of negative findings

The advantages of angiography are (*1*) it is easily done under local Xylocaine and intravenous Pentothal or Nembutal anesthesia; (2) good tolerance by the patient, (*3*) high degree of accuracy, (*4*) good visualization of vascular deformation despite increased intracranial pressure; (*5*) correct diagnosis in cases of simulated signs of head injury, as in a patient with a stroke or ruptured aneurysm who in falling strikes his head

Trephination

The diagnosis of intracranial extravasation of blood by means of burr openings in the skull is a time-honored method It is not infallible A subdural hematoma in the frontoparietotemporal area is usually discovered by diagnostic trephination An extradural hematoma, which may be at the base or more posteriorly in the parieto-occipital region, or near or at the midline, is harder to locate As a rule, burr openings are drilled at the frontoparietal junction, 3 to 4 cm from the midline on each side, at or near the coronal suture These often fail to reveal the hematoma, and multiple openings may be needed In some cases, burr openings near a fracture site may disclose an extradural hematoma Negative results of diagnostic trephination may mislead the surgeon When there is an intracerebral hematoma as well as a subdural collection, the latter is merely the overflow of the intracerebral clot Trephination, which fails to disclose the presence of an intracerebral hematoma, makes the surgical problem even more difficult

Bilateral trephination is indicated in all cases in which the presence of subdural hematoma is demonstrated, even though other procedures may suggest that the mass is unilateral

Although trephination is of value, particularly if ventriculography is performed, when the results of trephination are negative, angiography may be a more effective technic. Nevertheless, many surgeons still believe that diagnostic trephination is the method of choice.[11, 18 24, 27 31]

Pneumoencephalography (Figs 30 33)

This is a useful procedure in subacute head injuries and for patients with chronic complaints. In acute head injury particularly the first day or two, the procedure is often dangerous because of the possible presence of increased intracranial pressure, blood in the cerebrospinal fluid, or a forming mass lesion.[22]

Technic

The patient is placed in a sitting position, and anesthetized by 6 to 10 cc. of a 2 per cent Pentothal solution. By lumbar puncture, the cerebrospinal fluid is withdrawn and 80 to 120 cc. or more of air are injected By another method, the lumbar puncture is performed with the patient in the prone position, in order to record the cerebrospinal fluid pressure, immediately thereafter, the patient is placed in the sitting position. It is advisable to withdraw the cerebrospinal fluid completely unless this is done, the unequal distribution of air in the ventricles and subarachnoid spaces may give a false impression of cerebral abnormalities. As soon as the injection of air is completed, oxygen is administered to reduce nausea and vomiting, the patient is then placed in a special chair with chin supports, and roentgenograms in the upright and prone positions are taken. If no air has entered the ventricular cavities after 60 cc. or more have been introduced, a ventriculogram and surface exploration through skull openings may be performed. In some cases, the patient may need further study 8, 24, and 48 hours later During the injection of air, the patient's head should be moved back, forward, to the right, and to the left, so as to facilitate

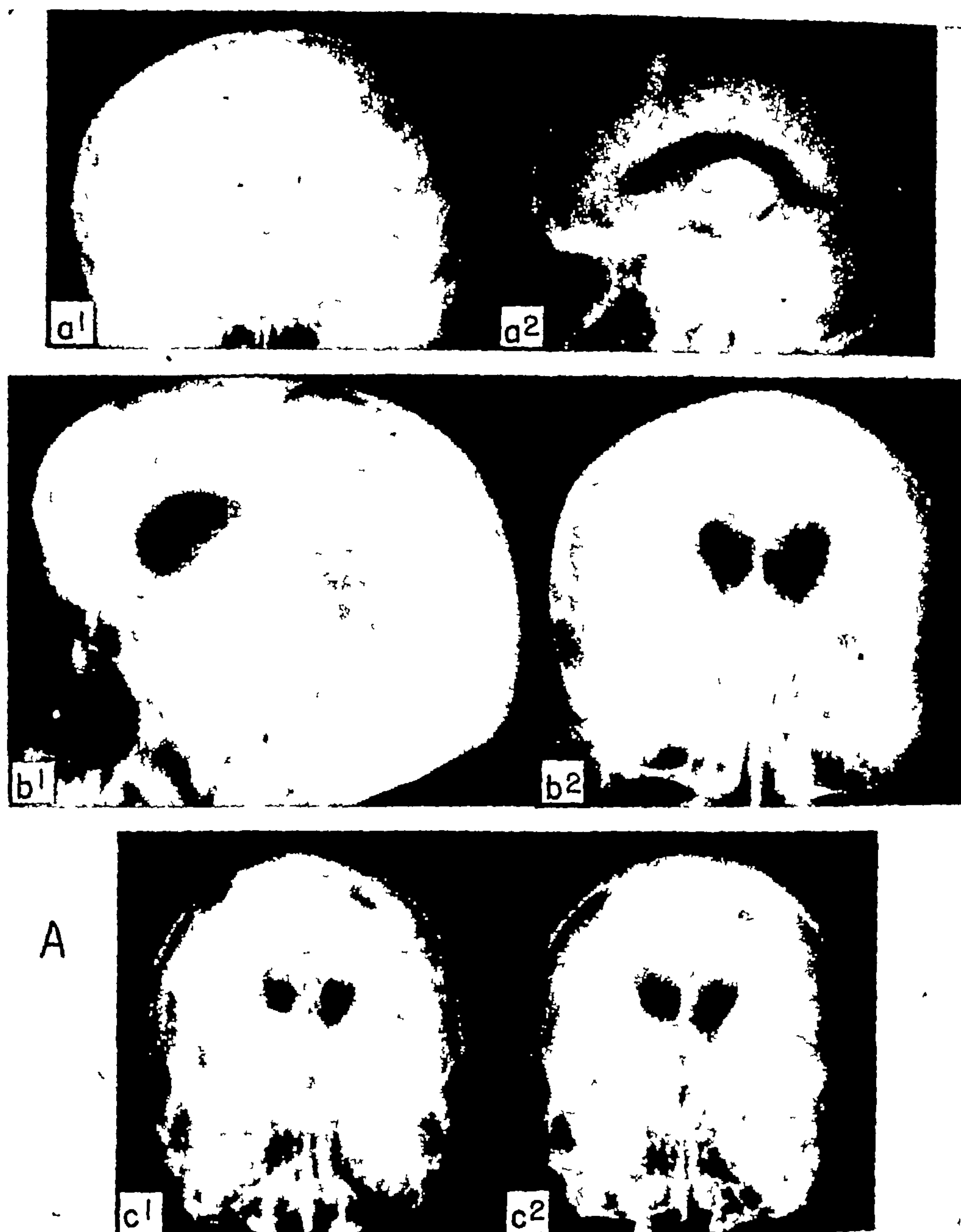


Fig 30 Pneumoencephalography in various craniocerebral lesions. (*A*) Normal pneumoencephalogram (*a*) of patient with cerebral palsy and bilateral spasticity. (*b*) Somewhat enlarged left ventricle and bilateral internal hydrocephalus in patient with left occipital skull fracture (*c*) Ventricular enlargement in 1 year and cerebral atrophy in patient with subdural hygroma

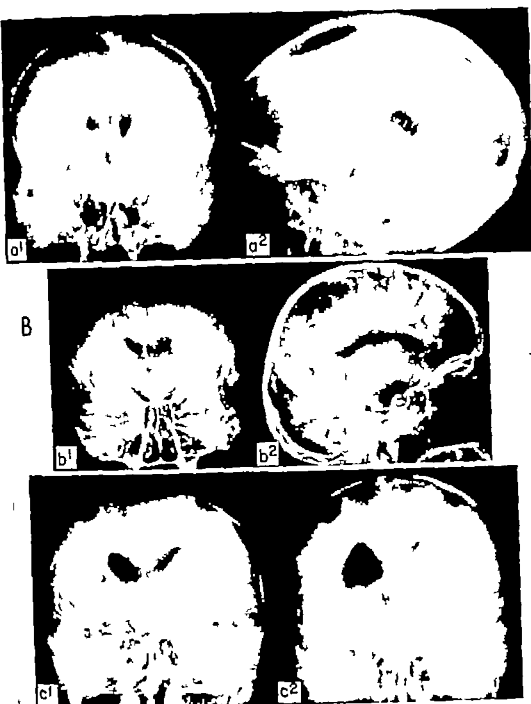


Fig. 30 (*continued*) (*B*) Ventriculogram (*a*) showing subdural cavity after evacuation of left subdural hygroma. (*b*) Cortical compression by left subdural hygroma. (*c*) Subdural hygroma, bilateral 1 year after drainage of hygromas (c^1) and 3 years later showing development of left frontal astrocytoma (c^2)

the escape of fluid and dislodge possible air bubbles which might prevent the passage of air into the ventricles or the subarachnoid spaces An occasional slight shaking of the head may also be helpful if the flow of fluid is insufficient

Findings

Chance plays a part in the pathways which the injected air follows Air enters the ventricular system of cavities through the foramens of Luschka and through the foramen of Magendie as well, and spreads into the fourth, third, and lateral ventricles Sometimes, the fourth and third ventricles are visualized, while the lateral ventricles are not In other cases, a bubble of air in the cerebrospinal fluid in a rather small foramen of Monro may prevent the entrance of air into the ventricles, in these cases, the patient should be allowed to rest for several hours, after which repeated roentgenography may show that air has entered the ventricles, with adequate visualization.

Atrophic and space-occupying lesions may involve various cisterns, including the cisterna magna, basalis, interpeduncularis, and chiasmatica, and the venae magnae cerebri Increased intracranial pressure will result in an encephalogram in which the ventricular outline is rounded superiorly and laterally in the anteroposterior view When this blunting of outline is combined with ventricular dilatation, it is unmistakable evidence of cerebral atrophy or increased intracranial pressure and hydrocephalus

Ventricular dilatation calls for careful evaluation of the cause Infarction due to a thrombosis of the internal carotid artery can cause extensive dilatation and hydrocephalus ex vacuo of one ventricle and possibly of the other ventricle as well In such a case, the ventricular enlargement antedated the head injury Cerebral atrophy may be suggested by the presence of increased fluid spaces in the subarachnoid areas The absence of air over a hemisphere may indicate a subdural collection of blood or fluid The injected air may collect under the

superior medial edges of a subacute or chronic subdural hematoma[9,10] (*see* Fig 62*B*), as Dyke[9] was the first to observe.

The encephalograms of a patient with bilateral mass lesions may be misleading, the side with the larger mass may be compressed and shifted to the opposite side, thus minimizing the abnormality which would otherwise be shown effectively

The absence of air on one or the other side over the cortex in children may or may not be significant. In many cases, exploration fails to reveal the cause of the abnormality Small amounts of fluid have been found over the cortex on the side without air in the subarachnoid spaces. Possibly, in such cases, a mechanical factor prevents the air from entering the subarachnoid spaces, or there may be abnormal connective tissue proliferation in the piarachnoid resulting from an old injury or inflammatory disease.

The diagnosis of cerebral atrophy by means of the encephalogram is complicated by many pitfalls (*1*) In an occasional case, air may enter the subdural space, so that the encephalogram gives the impression of cerebral atrophy, another encephalogram some time later will be completely normal. (2) In older persons, there is apparently some physiologic atrophy Other confusing factors are (3) Ventricular dilatation immediately after injury, it may be due to an increase of fluid in the ventricular cavities and subarachnoid spaces as a result of blood in the cerebrospinal fluid, or of increased fluid formation and decreased absorption. (*4*) Decrease in ventricular size soon after injury and enlargement after a year or more (Fig 30) (5) Localized ventricular enlargement, resulting from a hydrocephalus ex vacuo caused by a deep-seated cerebral contusion (Fig 31) (6) Ventricular dilatation in the vicinity of and extending toward a bone defect (Fig 32) (7) Ventricular dilatation in extensive cerebral trauma (Fig 31), as in penetrating wounds.

In summary, the causes of enlargement of the ventricles and subarachnoid pathways are (*1*) an increase in the volume of cerebrospinal

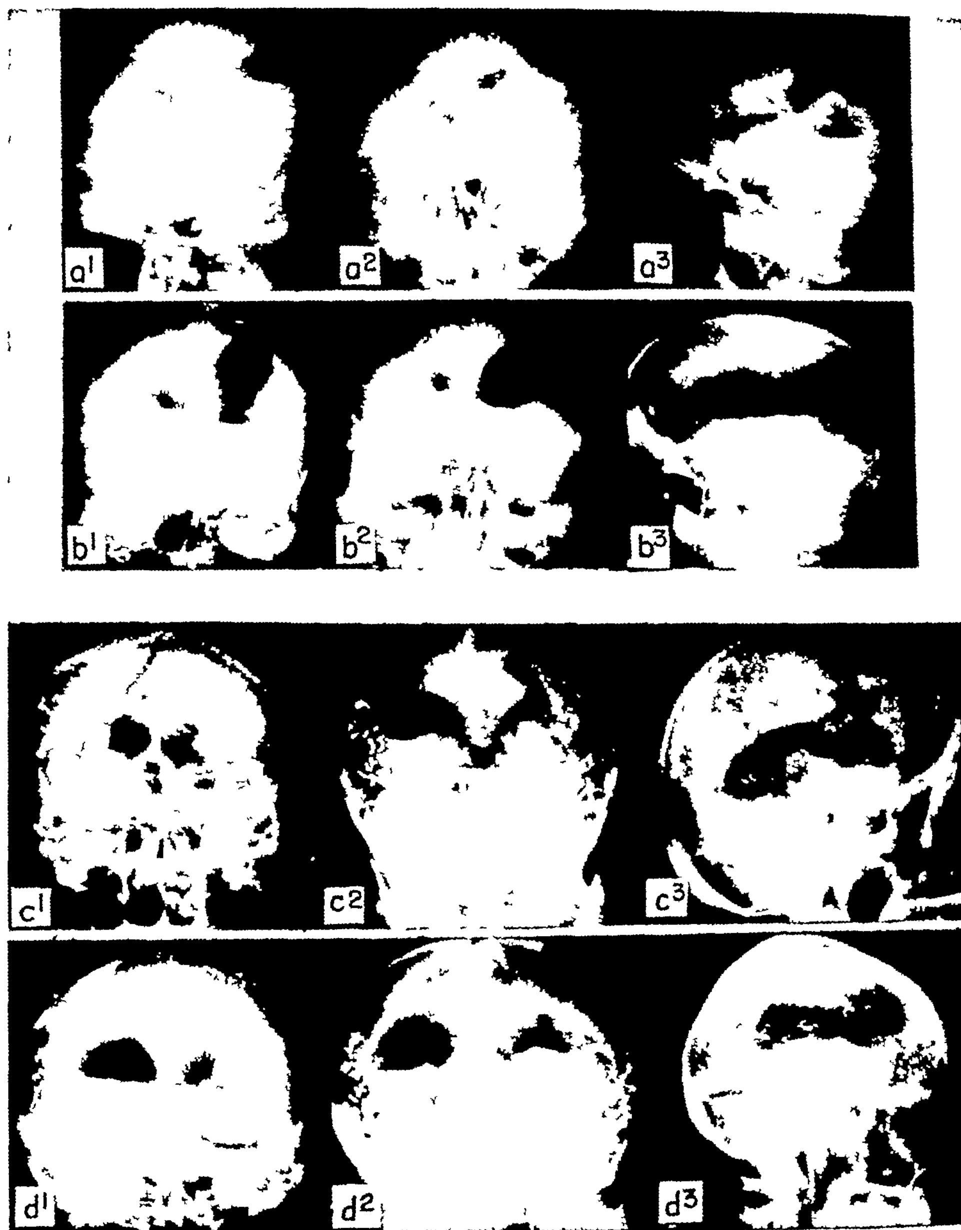


Fig 31 Pneumoencephalography in penetrating head injury (*a–b*) Pneumoencephalograms 38 days after insufficient primary debridement and 41 days later after secondary debridement, respectively marked enlargement of ventricles and of porencephalic cyst (*c–d*) Pneumoencephalograms 14 days after primary debridement and 57 days after secondary debridement, respectively, marked enlargement of right ventricle.



Fig. 32. Pneumoencephalography in head injury (*a*) Pneumoencephalograms 2 years after skull fracture with extensive cerebral destruction hydrocephalus ex vacuo, particularly large on right side no air over hemispheres patient bedridden, with aphasia and bilateral spasticity more marked on left side. (*b*) Pneumoencephalograms 10 years after head injury suffered by 8 year old child large porencephalic cyst in left occipital area, communicating with posterior horn of left lateral ventricle (b^2) extensive bone absorption over lesion right homonymous hemianopsia posttraumatic epilepsy (*c*) Enlarged ipsilateral ventricle after open skull fracture hydrocephalus ex vacuo mild posttraumatic epilepsy



Fig 33 Pneumoencephalography in head injury (*a*) Head injured in fall from horse, hematoma in midline between hemispheres, compressing ventricular cavities (*b*) Left subacute subdural hematoma, 8 days after injury, left ventricle definitely compressed, less air on left side (*c*) Right subacute subdural hematoma; right ventricle somewhat compressed, no air in subarachnoid pathways on right. (*d*) After evacuation of liquid subdural hematoma surrounding hemisphere in infant.

fluid, (2) inadequate absorption of cerebrospinal fluid, (3) blocked circulation of cerebrospinal fluid as a result of contusions, hemorrhages, etc., and (4) cerebral contusions and atrophy, with hydrocephalus ex vacuo.[1,3,6,14,17,24,25,27,28,35]

Ventriculography

When a diagnostic air study is indicated in the acute phase of a head injury, ventriculography is a more advisable procedure than lumbar pneumoencephalography As a rule, ventriculography is combined with bilateral exploratory trephination in the frontoparietal areas. If the latter procedure does not reveal a subdural collection of fluid or blood, it is safe to introduce a brain cannula, proceed with the ventricular tap, and introduce air Elective ventriculography may be possible in some cases, it is best done through openings in the parieto-occipital area, with the patient in a sitting position. Occasionally, a chronic subdural hematoma may be identified through such openings in the skull.

Technic

It is important that the ventricle be entered with one stroke of the needle, several punctures permit escape of the injected air into the subdural or subarachnoid spaces, particularly the former, with resultant poor visualization of the ventricles. Bilateral injection of air assures better filling To accomplish this, 30 cc. or more of air are injected into one ventricle while a stilet is in place in the other, the needle is swiftly withdrawn as soon as the injection is completed, and the maneuver is repeated on the other side. This method results in better visualization than by the one in which air is introduced by the substitution method The air should not be injected under pressure, but complete filling should be sought. Movement of the head helps to fill the ventricles completely if the patient is in the supine position, the surgical draping should allow the head to be moved to the right and left, if the patient is sitting, the head is hyperextended The vital functions must be carefully watched throughout the procedure.

Cerebrospinal Fluid Studies

A lumbar puncture, or a cisternal one if the lumbar puncture cannot be made because of skeletal or spinal fracture, is of diagnostic value in many cases of head injury. Routine lumbar puncture for diagnosis or treatment is inadvisable For diagnosis it should be used when there are definite indications for it; therapeutic puncture must be resorted to cautiously and on an individual basis In the early stages of a head injury, a lumbar puncture may affect the intracranial hydrodynamic status and so disguise possible signs and symptoms of a dynamic lesion, *e.g.*, a forming extradural hematoma Repeated withdrawal of fluid in such a case would tend to keep the cerebrospinal fluid pressure from increasing, and the patient could die because the lesion had not been recognized in time.

The following data are important in cerebrospinal fluid studies. (*1*) pressure, (2) blood content and cell count, and (*3*) protein content In our experience, a fluid initially very bloody or grade 4 clears in 8 to 10 days, and in some cases becomes only faintly xanthochromic within 2 days. The red blood cells hemolyze and disappear from the fluid much more rapidly than the leukocytes, so that puncture after 4 or 5 days may reveal a xanthochromic fluid with a leukocyte count out of proportion to the red blood cell count (normal red to white blood cell ratio, about 400.1) The leukocytes are about equally divided between polymorphonuclear cells and lymphocytes A high protein content is considered to be diagnostic of cerebral damage and implies a poor prognosis, although the value of diagnostic lumbar puncture has been emphasized in patients with serious injury, we have not found a high protein level to be a consistent feature of such injury The findings in 411 patients of our series of 1,285 cases are shown in Table 3 The variations in mortality rate with the type of fluid found are obvious

While the cerebrospinal fluid pressure does not invariably reflect accurately the intracranial fluid pressure, studies by various investigators

of the changes in pressure under different conditions have established a good correlation in many cases.[3, 4, 13, 16, 23a, 36, 38]

TABLE 3 *Cerebrospinal Fluid Findings in 411 Cases of Head Injury*

Finding	*Number of cases*	*Number fatal*	*Mortality rate*
Pressure > 200 mm. H_2O	187	38	20.3
Bloody	195	54	27.6
Xanthochromic	55	9	16.3
Clear	161	6	3.7
Protein > 60 mg./100 cc.	81	14	17.2

Several types of cerebrospinal fluid hypotension have been described (*1*) hypotension caused by a partial or complete block as a result of herniation through the dural compartments, *e.g.* tentorial or foramen magnum herniation, (2) primary hypotension in a seriously ill patient caused by general relaxation of the arterial walls, (*3*) hypotension as part of the postconcussion syndrome. The presence of a partial or complete block may be revealed by the Queckenstedt maneuver, but this test may be dangerous in a patient with a severe intracranial lesion. The result of the test in primary hypotension is normal. The low cerebrospinal fluid pressure in these cases may be associated with a high pulse pressure. The cerebrospinal fluid hypotension of the postconcussion state resembles that which sometimes occurs after lumbar puncture. In both, the headache which it causes is relieved by lying down. The hypotension after lumbar puncture may be the result of leakage of fluid via the needle path into the subdural or epidural region. A low pressure does not rule out the presence of an intracranial mass lesion, but with most dynamic lesions the pressure is fairly high — usually over 300 mm. of water.

Electroencephalography (Figs. 34-40)

The classification of the electroencephalographic features of head injury used here is a modification of the one set up by Bickford and

Whelan.[3] Abnormal records are divided into two broad categories rhythmic abnormalities or dysrhythmias, and arrhythmic abnormalities, or arrhythmias or deltas. The term "delta" is used in its original meaning of arrhythmic, nonrepetitive, slow waves of less than 4 cycles per second While this classification might lead to some confusion, since delta, strictly speaking, is also used for any slow wave of a lower frequency than 4 cycles per second, there are clinical advantages in separating out those records which show arrhythmic slow waves Thus, an EEG showing slowing to 2 cycles per second is classified as dysrhythmia, whereas one showing arrhythmic slowing to 2 cycles per second is classified as delta

The details of the classification are as follows

Dysrhythmia (also characterized as focal or diffuse) General category for waves which are usually rhythmic, repetitive, tending to be inhibited by opening of the eyes, and accentuated by hyperventilation Specific wave forms are also included in this category

Grade I Minimal continuous or paroxysmal rhythmic slowing; amplitude up to 30 μv

Grade II Moderate continuous or paroxysmal rhythmic slowing, amplitude up to 60 μv

Grade III Specific wave forms, *e g*, spikes, sharp waves, spikes and waves

Delta (also characterized as focal or generalized) Random, slow waves (0 5 to 4 per second), arrhythmic and nonrepetitive, usually unaffected by opening of the eyes or by hyperventilation

Grade I Amplitude up to 30 μv

Grade II Amplitude between 30 and 60 μv

Grade III Amplitude above 60 μv

Asymmetry Amplitude asymmetry of the alpha rhythm greater than 25 per cent

Changes in Specific Conditions

In severe head injuries, in which there is extensive cerebral damage, contusion, and hemorrhage of varying severity, the EEG is not particularly useful in the first day or two after injury. The waves from the scalp surface may show only a slight change from normal, or there

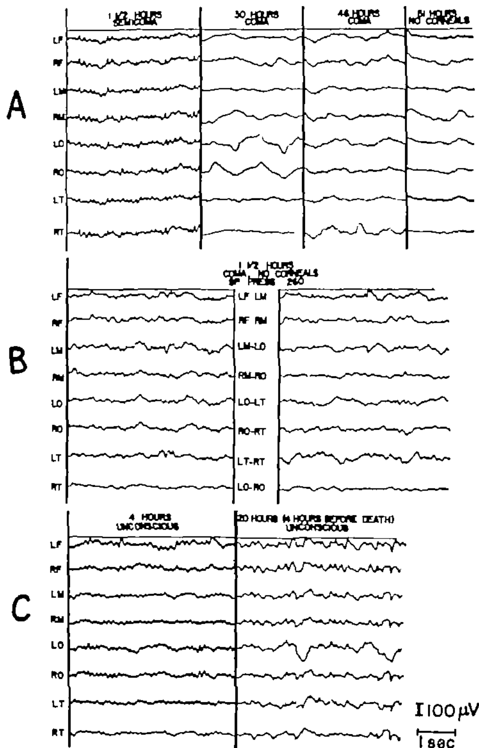


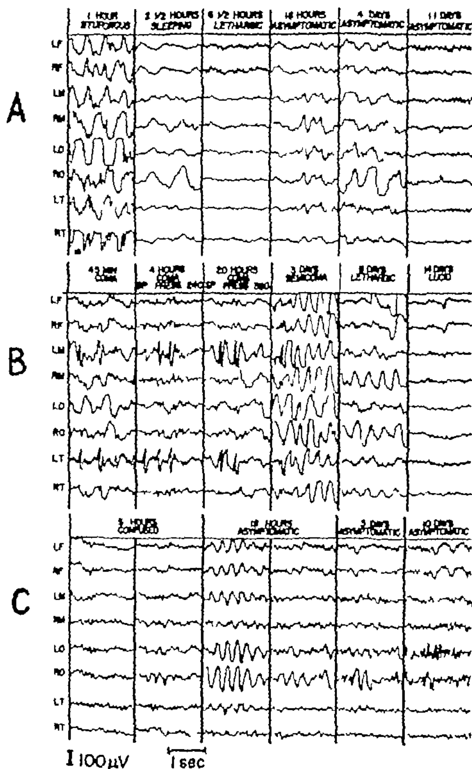
Fig 34 Electroencephalography in head injury (SP PRESS. cerebrospinal fluid pressure in millimeters of water) (*A*) Right temporal and basal fracture patient dead 71 hours after injury autopsy showed thin subdural hematoma on right, small, right intratemporal lobe hemorrhage, and small softened areas in left frontal lobe at 1½ hours, EEG almost normal progressive abnormalities thereafter (*B*) Patient, 48 years old, dead 2 hours after injury autopsy showed massive subdural hematoma on left EEG gave no indication of mass lesion. (*C*) Patient, 36 years old, dead 24 hours after gunshot injury· entrance wound in left parietal area, exit through right motor cortex at 4 hours, EEG comparatively normal at 20 hours, EEG shows considerable deterioration more marked on left.

may be marked damping, with unusually low voltages.[7] Thereafter, as the patient's state stabilizes, more obvious abnormalities become evident (Fig 34) The early low-voltage waves may be due to involvement of the reticular formation of the brain stem by hemorrhage, or to a dysfunction in this region without gross lesions

The EEG in less serious injuries without a mass lesion may be normal or only slightly abnormal immediately after the injury, it usually begins to show marked abnormalities over a period of several days, without necessarily correlating with the clinical status

In the presence of extradural or subdural hematoma (Figs 33 and 34), the electric emissions from the cerebral surface are often blanketed, resulting in a definite lowering of the electric potential on the affected side With a bipolar method, the blanketing effect may be due to a short-circuiting of the cortical potentials in the blood clot, which is a good conducting medium[18, 31] The low-voltage waves which may appear for a day or two after the clot is removed might be explained on the basis of a subdural collection of cerebrospinal fluid, but since the same effect is seen when a drain is used, in which case there is no subdural collection of fluid, the short-circuiting theory does not seem to be a satisfactory explanation In some cases, delta foci, like those found in patients with brain tumors, may be seen, but they often appear late in the course of the lesion[21, 23, 32, 40]

Fig 35 Electroencephalography in acute head injury in children (SP PRESS: cerebrospinal fluid pressure in millimeters of water) (*A*) Right temporal bone fracture in 3 year old girl; unconscious for 5 minutes, several months after injury, normal EEG, at 1 hour after injury, convulsive activity with grade II delta waves; at 2½ hours, decrease in electric activity, at 6½ hours, flattened waves, suggesting lowered activity of reticular formation by edema, at 18 hours and 4 days, abnormalities still present (*B*) Head injury without skull fracture in 6 year old girl, unconsciousness for 10 minutes, aphasia, and right facial paresis for 2 weeks; at 45 minutes after injury, EEG shows grade II dysrhythmia and grade I delta waves more pronounced on left, at 4 hours, spike activity from left motor and temporal areas, at 20 hours, persisting spike activity and general dysrhythmia, at 8 days delta activity on right rather than on left, at 14 days, EEG fairly normal (*C*) Head injury without fracture in 7 year old girl, drowsiness and confusion for 8 hours, asymptomatic after 18 hours, at 5 hours after injury, EEG shows slight abnormalities at 18 hours, definite abnormalities, at 10 days, some abnormalities persisting despite absence of symptoms


A
1 HOUR
6 1/2 HOURS
LETHARGIC
ASYMPTOMATIC
ASYMPTOMATIC
ASYMPTOMATIC
B
LETHARGIC
C
ASYMPTOMATIC
LF
RF
LM
RM
LO
RO
LT
RT
100µV
1 sec

In subacute and chronic subdural hematomas, the EEG accurately localizes or lateralizes the lesion in only 45 per cent of cases, in our experience The EEG often shows bilateral involvement, although the hematoma may be unilateral.

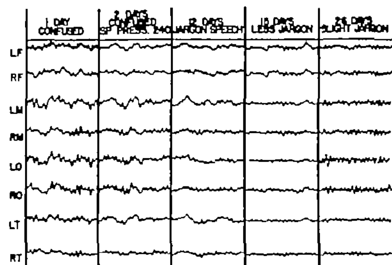
The EEG in the posttraumatic cerebral syndrome of an injury without initial unconsciousness is often slightly disorganized; in many cases, however, it is normal Occasionally, there is an abnormality on the side of the blow, and also at the site of the headache Sometimes, the patient continues to complain, although the EEG has become normal Correct evaluation of posttraumatic states may require repeated electroencephalographic examination

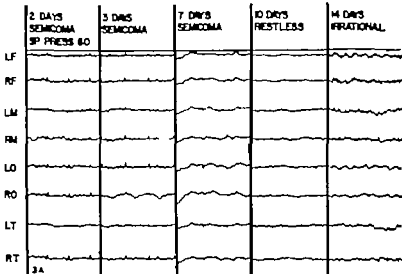
In posttraumatic states with an initial period of unconsciousness of several minutes to several hours, the EEG may at first show disorganization, but soon improves, along with the complete or almost complete disappearance of symptoms In posttraumatic states with aphasia, paresis, or paralysis due to involvement of the motor cortex, the patient's status may remain unaltered although the EEG may show progressive improvement (Figs. 35-36).

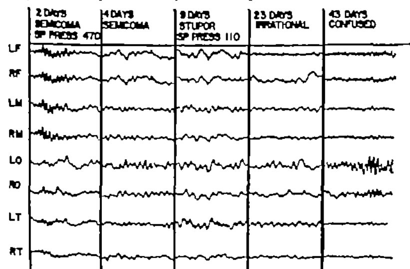
The patient's age is an important factor in the evaluation of posttraumatic abnormalities of the EEG They are found more frequently in the younger age groups than in the older ones. Diffuse abnormalities, it has been suggested, may represent constitutional defects, while focal ones probably represent true lesions of traumatic origin. Once the acute phase of a head injury is past, it is wiser not to ascribe a

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Fig 36 Electroencephalography in head injury (SP PRESS cerebrospinal fluid pressure in millimeters of water) (*A*) Left temporal bone fracture and contusions of left motor and temporal areas in 50 year old man, unconscious for 1 hour, expressive aphasia for 8 weeks, at 26 days after injury, EEG shows no abnormality in left motor area. (*B*) Fracture of right frontoparietal area in 55 year old man, unconsciousness for 12 hours, incomplete recovery during observation period; EEG shows considerable artifact, no marked changes, indicating possibility of brain stem injury. (*C*) Bilateral parietotemporal fractures in 29 year old man, unconsciousness for 24 hours, angiograms negative, at 2 and 4 days after injury, EEG shows dysrhythmia and grades I and II delta waves, at 9 and 23 days, no change in slow activity, no apparent correlation between clinical picture and EEG.


100µV
1 sec
A
1 DAY
CONFUSED
2 DAYS
SP PRESS. 240
JARGON SPEECH
LESS JARGON
SLIGHT JARGON
LF
RF
LM
RM
LO
RO
LT
RT


B
2 DAYS
SEMICOMA
SP PRESS 60
3 DAYS
SEMICOMA
7 DAYS
SEMICOMA
10 DAYS
RESTLESS
14 DAYS
IRRATIONAL
LF
RF
LM
RM
LO
RO
LT
RT
3A

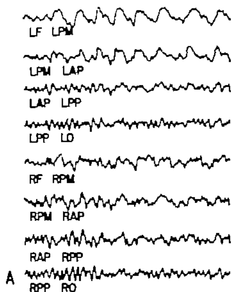

C
2 DAYS
SEMICOMA
SP PRESS 470
4 DAYS
SEMICOMA
9 DAYS
STUPOR
SP PRESS 110
23 DAYS
IRRATIONAL
43 DAYS
CONFUSED
LF
RF
LM
RM
LO
RO
LT
RT

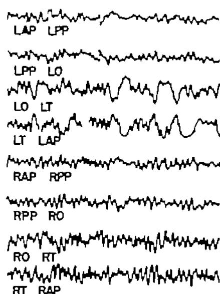
generalized abnormality to the injury; this may be of considerable importance in the medicolegal aspects of a head injury It has been pointed out that a generalized EEG abnormality 2 years or more after injury is no more or only a little more significant than it would be were there no history of head injury, a focal abnormality, on the other hand, is most probably the result of the injury and correlates well with the site of the injury and the occurrence of focal seizures and residual neurologic signs [16] Nevertheless, focal abnormalities may disappear despite the continued presence of neurologic deficits (Fig 36)

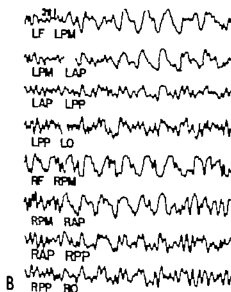
Paroxysmal outbursts in the EEG, including rhythmic high-voltage waves of 2 to 3 per second, fast rhythms of 22 per second, and outbursts of intermediate frequency, are a definite indication of the likelihood of posttraumatic epilepsy

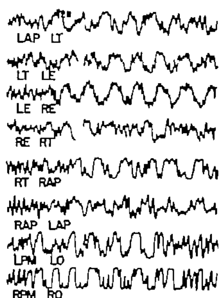
We have studied, with Dr Whelan's help, the effects on the EEG of photic stimulation in 12 patients with head injuries of varying severity Although so far it is difficult to ascribe any special significance to the finding that such stimulation affected the posttraumatic EEG, it may possibly prove to be an important diagnostic and prognostic aid in serious head injury Involvement of the reticular formation and of the upper brain stem may be so severe that incoming photic stimuli may fail to register and so cause no electroencephalographic change A local injury to one or the other optic pathway cannot explain the lack of response to photic stimulation to be seen in Figure 39; a more likely explanation is involvement of an area where both pathways may be equally affected, for example, depression of the pace-setter mechanism

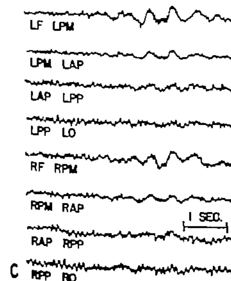
Fig 37. Electroencephalography in head injury, patient semicomatose and disoriented on hospital admission after fall, subacute subdural hematoma removed 13 days after injury. (*A*) At 7 days after injury, EEG shows random and rhythmic slowing in frontal and temporal areas, more marked on left, patient conscious and alert, but with generalized headaches (*B*) At 12 days, bilateral rhythmic slowing in frontotemporal area and increased arrhythmic slowing in left temporal area (rhythmic slowing is projected abnormality, probably due to displacement of mid brain or brain stem, arrhythmic slowing is due to direct cortical involvement). (*C*) At 23 days, cortical activity somewhat restored.

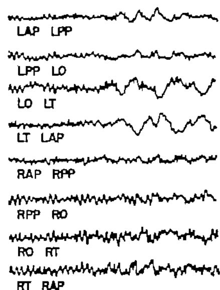

LF LPM
LPM LAP
LAP LPP
LPP LO
RF RPM
RPM RAP
RAP RPP
A
RPP RO


LAP LPP
LPP LO
LO LT
LT LAP
RAP RPP
RPP RO
RO RT
RT RAP


LF LPM
LPM LAP
LAP LPP
LPP LO
RF RPM
RPM RAP
RAP RPP
B
RPP RO


LAP LT
LT LE
LE RE
RE RT
RT RAP
RAP LAP
LPM LO
RPM RO


LF LPM
LPM LAP
LAP LPP
LPP LO
RF RPM
RPM RAP
1 SEC.
RAP RPP
C
RPP RO


LAP LPP
LPP LO
LO LT
LT LAP
RAP RPP
RPP RO
RO RT
RT RAP

The electroencephalographic pattern of birth injuries is difficult to evaluate, particularly because of the marked variability in the tracings of infants and children and the lack of definite knowledge of the normal range in this age group. It has been suggested that prenatal and birth trauma may cause irreversible damage[16] But even in this group, maturation, with a tendency to improvement, does occur. If cerebral tissue is locally destroyed, delta activity, commonly seen up to the age of 6 months, may persist Thereafter, when normally the delta waves disappear rapidly and theta and sometimes alpha rhythms begin to appear, silent areas due to agenesis, or delta rhythm due to dystrophy or developmental arrest, may be recognized in the EEG Repeated examinations may show the persistence of the delta

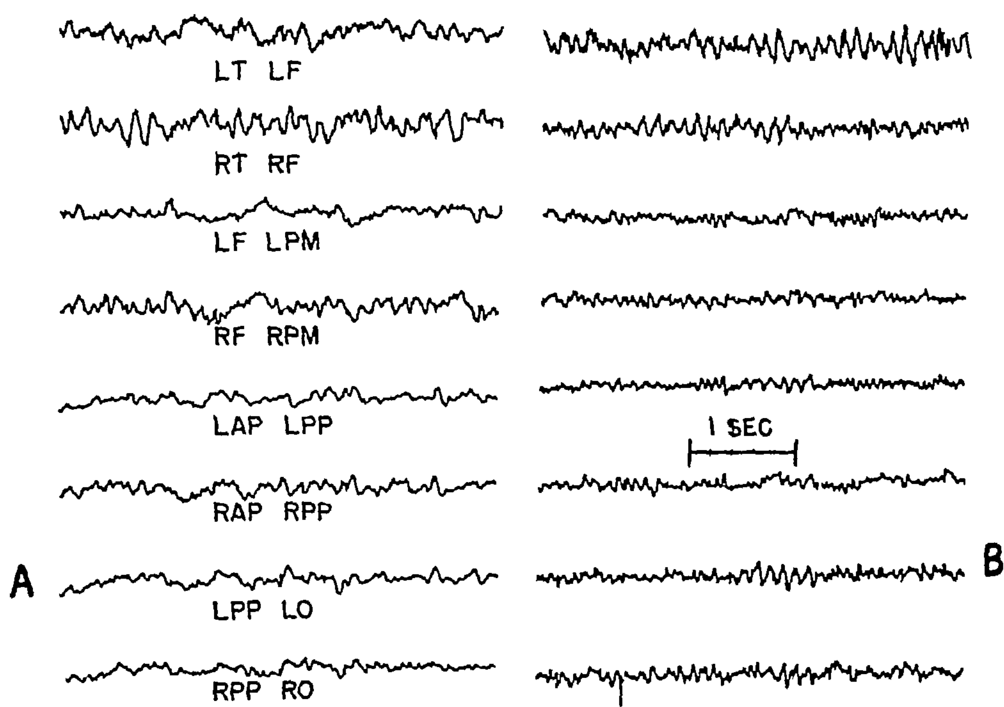


Fig 38 Electroencephalography in head injury acute s… …oma, patient semiconscious on hospital ad… …ion, aphasia, ri… …gia, rapidly worsening condition, removal … …atoma follc… …pearance of paralysis in a few days and c… …ut 6 weeks (… (30 minutes before angiography ar… EEG shows s… …ied waves from left frontal, premotor, … (B) At 4 … normal, patient asymptomatic

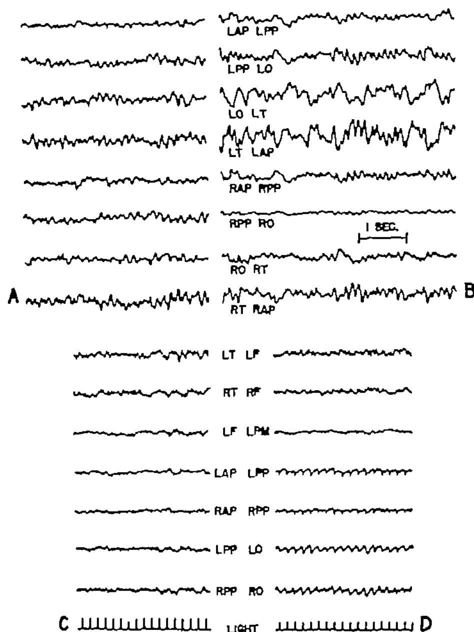


Fig 39 Electroencephalography in head injury patient, 40 year old man comatose and with alcoholic breath on hospital admission. (*A*) At 1 hour 35 minutes after injury EEG shows generalized grade II dysrhythmia, maximal in temporal, parietal, and occipital areas greatest slowing in left temporal area. (*B*) At 30 hours, generalized grade II dysrhythmia maximal slowing and grade I delta waves in left temporal area. (*C*) Photic stimulation at 6 days without effect, patient still lethargic. (*D*) At 10 days, grade I delta waves and grade I dysrhythmia in left temporal area evidence of driving on photic stimulation patient alert

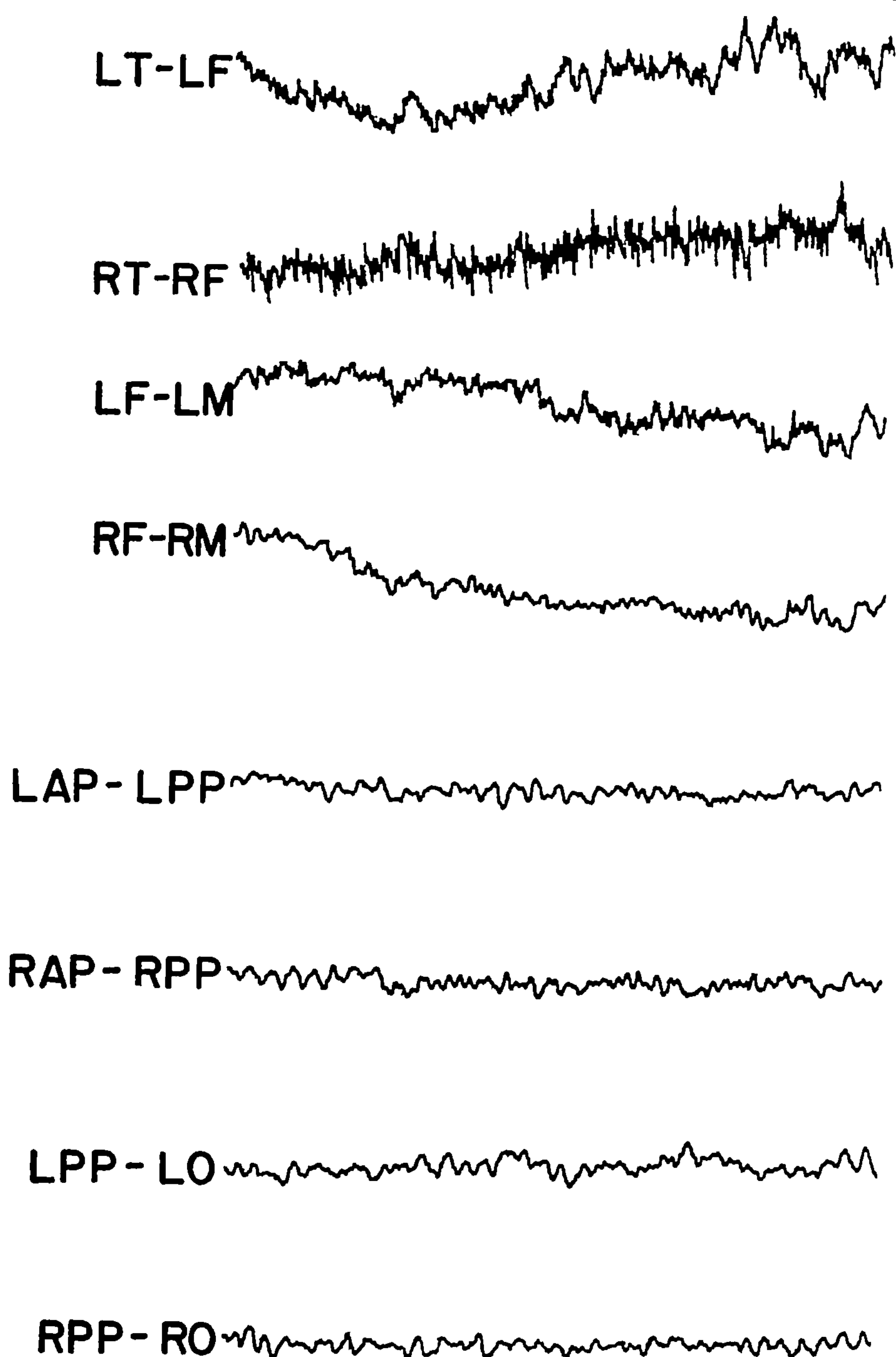


Fig 40 Electroencephalography in head injury, patient, 63 year old woman, fell and struck back of head, chronic subdural hematoma At 2½ months, EEG shows grade I delta waves in left temporal area and bitemporal grade I dysrhythmia, more pronounced on left Pneumoencephalogram showed a mass in left parieto-occipital area, operation revealed large, almost completely solid hematoma, without temporal extension

rhythm or failure of this rhythm to be replaced by a theta or an alpha component, indicating the presence of definite functional impairment. If the impaired function is in a silent cortical area, the EEG or the pneumoencephalogram may be the only means for correctly assessing the lesion.

In summary the EEG seems to be of little diagnostic value in the early stages of a serious head injury, in subacute and chronic states it is of greater value. Mass lesions may cause a damping of the voltage on the affected side, and in subacute or chronic cases there may be focal delta activity Bilateral abnormality suggests an involvement of the pace-setter mechanism in the reticular formation The electroencephalographic findings in the case of a person who has suffered a mild injury but complains of posttraumatic sequelae, or has clinical symptoms, range from normal through a gamut of abnormalities It is also possible for the EEG to be abnormal although the patient is apparently well. One of the great difficulties in evaluating minor electroencephalographic changes in this group of patients is the absence of pre injury records.

From our data on 57 cases of postconcussion syndrome, 64 cases of postsubconcussion syndrome, and 200 cases of headache without neurologic deficits, we have found no significant electroencephalographic abnormalities which differentiate the groups from each other

Radioactive Isotope Studies

No definite conclusions can be drawn so far from the few available reports on the use of radioactive isotopes for localizing traumatic and nontraumatic cerebrovascular lesions. Dumbar and Ray[8] using radioactive iodinated serum albumin (RISA), correctly located a subdural hematoma in 4 cases, the radioactivity was 20 to 30 per cent higher on the side of the lesion, but the clot itself showed no uptake of the radioactive material Rushton and co-workers[20] were able to locate subdural hematoma in 2 of 3 cases, in 1 of the 2, with bilateral hematomas, the

uptake was higher on one side than on the other, in the third case, with a 100 cc liquefied clot, there was no localization with RISA Sweet and Brownell,[34] using radioactive arsenic (As^{74}), correctly diagnosed 2 of 4 cases of subdural hematoma In 3 of our cases of acute intracerebral hematoma in which we have used RISA, there was no significant difference in uptake on the two sides in 1 case, the localization was correct in 1 case, and on the wrong side in the third case

As yet, the localizing value of isotopes, as shown by studies with RISA and As^{74}, has not been outstanding No doubt, as experience with larger series of cases increases, a more accurate estimate of this diagnostic technic in head injury will become possible.

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Chapter V

CLINICAL MANIFESTATIONS OF HEAD INJURY

Injury of the head may be mild, and without immediate or late effects, or it may be so severe that it is fatal within minutes A short, accurate history should be obtained in each case, if at all possible, it is essential for the evaluation of the injury A story of a fall, a blow on the head, or that the patient became dizzy or unconscious and then fell may be important A history of alcoholism or an alcoholic breath should be noted The possibility of multiple lesions should always be kept in mind, the patient with a head injury may also have injuries in the chest, abdomen, extremities, spine. The patient must be regarded as a whole, not in strictly separated compartments In the early stages, the patient's condition should be checked frequently in order to note the appearance of symptoms and signs which had not been present at first

The examination in the early stages of an acute, severe head injury must often be limited because the patient is unable to co-operate Certain basic observations, however, should be made The responses to stimuli, a clue to the degree of unconsciousness, should be carefully evaluated and recorded A record should be made of the state

of the vital functions, this is important in evaluating shocklike states. In addition, the scalp should be inspected for injuries contusions and lacerations, the state of the pupils, corneal reflexes and fundi is noted, and the presence or absence of bleeding from any body orifice of any

TABLE 4. *Data on 1,285 Cases of Acute Head Injury 1952–1954**

Feature	*Number of cases*	*Number fatal*	[illegible]	*Mortality rate of 144 fatal cases* %
Distribution by age group				
0–10 yr	201	10	0.8	6.9
11–20 "	116	5	1.4	3.5
21–30 "	249	20	1.6	13.9
31–40 "	217	22	1	15.3
41–50 "	202	21	1.6	14.6
51–60 "	154	25	1.9	17.4
61 and over	143	41	3.2	28.5
State of consciousness				
Unconsciousness before hospital admission	539	5	0.9	3.5
Continued unconsciousness	299	117	38.1	81.3
Unconsciousness after admission (lucid interval)	21	14	66.7	9.7
No loss of consciousness	426	8	1.9	5.6
Time of death after hospital admission				
0– 5 hr	—	11	0.9	7.6
6–10 "	—	26	2.0	18.1
11–15 "	—	17	1.3	11.8
16–24 "	—	17	1.3	11.8
1– 2 days	—	12	0.9	8.3
3– 5 "	—	29	2.3	20.1
6–10 "	—	20	1.6	13.9
11 or more	—	12	1.9	8.3

*Sex 1015 male, 270 female. Skull fracture present in 637 (mortality rate, 17.2 per cent) no skull fracture in 648 (mortality rate 5.8 per cent)

gross abnormality of motor and sensory functions and of reflexes, and of injuries elsewhere in the body (particularly the cervical spine) should be ascertained There may be nuchal rigidity in patients with bloody cerebrospinal fluid, or limpness or flaccidity with a generalized areflexia in a profoundly ill patient, the latter is an ominous sign

Table 4 summarizes certain findings in our series of 1,285 cases of acute head injury seen between 1952 and 1954

State of Consciousness

Unconsciousness may be defined as an inability to respond to external stimuli in a manner intelligible to oneself and to the observer. Disturbances of consciousness range through drowsiness to a clouding of consciousness, with confusion and disorientation, to coma, with loss of some or all the reflexes These disturbances may be associated with restlessness and violent hyperkinesias

The mechanisms of unconsciousness after head injury are still incompletely understood From experimental evidence (*see* Chapter III), it seems a justifiable conclusion that cellular changes, with chromatolysis and injury of nerve fibers in the reticular formation of the brain stem and in the midbrain, cause the posttraumatic unconsciousness in blunt head injury Apparently, neither anoxia nor chemical or metabolic changes play a role[26] Were it possible to injure the entire cortex by a blow to the head, the effects would be the same as that of reticular formation involvement, *i e.,* unconsciousness However, in blunt head injuries, the anatomy of the head facilitates derangement of brain stem centers rather than of all cortical mechanisms The conscious state, of course, is also affected by contusions and lacerations of the higher centers Thus, with injury of the visual and speech centers, or of the frontal and limbic lobes, the patient's ability to assess the incoming afferent impulses is impaired Akinetic mutism results not only from involvement of the brain stem centers but also from an injury of the entire cortex The aphasic state and abnormalities of behavior after

lobectomy are evidence of the effect of supranuclear lesions upon the basic conscious state.

Derangements of the conscious state are of diagnostic importance. In acute injury, a normal consciousness is a favorable sign and a contraindication to immediate surgical intervention except in patients with open wounds and skull defects. Progressive improvement in the state of consciousness is also a good sign and almost always a contraindication to surgical intervention. On the other hand, increasing deterioration of consciousness, particularly with an intervening period of lucidity, is a sign that a dynamic lesion — hemorrhage extradural, subdural, or intracerebral hematoma, subdural hygroma, cerebral edema — may be present, and surgical intervention may be necessary. The same picture may occur with localized contusions, particularly when they involve the hemispheral convexities.

Extreme disorientation, alternating with periods of semiconsciousness and psychotic manifestations, may occur in injury to the tips of the temporal lobes, and to Brodmann's area 45 at the junction of the frontal and temporal lobes.

Generally, a patient who has been unconscious for many days regains consciousness gradually, with intervening periods of semiconsciousness, disorientation, and confusion. Only occasionally is there a prompt return to consciousness.

A closed head injury with prolonged unconsciousness has been reported to be accompanied by a greater incidence of epilepsy, but with a longer interval between the injury and the appearance of the epilepsy than in open head injuries.[50] We have not found this to be the case.

Amnesia

At least 80 per cent of patients with posttraumatic unconsciousness have a retrograde amnesia for the period immediately preceding the injury and for the accident producing the injury. In some cases, the amnesia may not be apparent because the patient responds satisfac

torily directly after the accident and for some time thereafter. These patients may have had an extremely short period of unconsciousness, or a dazed feeling for only several seconds if the blow was of subconcussive force The so-called posttraumatic automatism[10] is characterized by relatively normal behavior and responsiveness for a period which the patient then totally forgets when he regains complete consciousness In such cases, there may be contusions or lacerations of the base of the frontal lobes or the medial aspect of the temporal lobes, and involvement of the brain stem centers

Vital Functions (Fig. 41)

The effects of head injury on the vital functions in the experimental animal are discussed in detail in Chapter III

Pulse

In severe head injury, the pulse may initially be full, slow, and bounding, and become faster as improvement sets in In the absence of improvement, the pulse rate grows even slower, but eventually increases suddenly in rate and decreases in amplitude Thus, a pulse rate of 40 to 55, quite common in acute head injury, may in the course of 10 to 24 hours increase to 120 to 180 or higher, and then become thready if the injury is fatal

In some patients who have not lost consciousness and who are feeling quite well, the pulse rate may drop to 40 or 50 The low rate may be due to a period of vagal stimulation, with a resultant bradycardia; it usually subsides in 2 or 3 days In others, the pulse rate may be within the normal range initially, but in the course of several days decrease When accompanied by other signs of increasing intracranial pressure, the presence of a dynamic intracranial lesion is indicated In still others, the pulse rate may be fast from the very beginning and be associated with a shocklike state, particularly if there are injuries to other parts of the body.

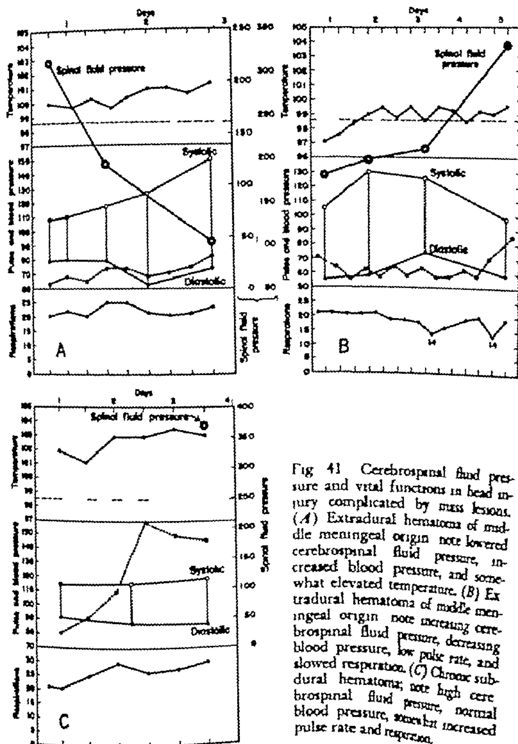


Fig 41 Cerebrospinal fluid pressure and vital functions in head injury complicated by mass lesions. (*A*) Extradural hematoma of middle meningeal origin note lowered cerebrospinal fluid pressure, increased blood pressure, and somewhat elevated temperature. (*B*) Extradural hematoma of middle meningeal origin note increasing cerebrospinal fluid pressure, decreasing blood pressure, low pulse rate, and slowed respiration. (*C*) Chronic subdural hematoma; note high cerebrospinal fluid pressure, normal blood pressure, somewhat increased pulse rate and respiration.

Respiration

In slight head injury, the respiration may be unaffected In serious injury, respiration may be (*1*) fairly normal at first, then slowed, suggesting the development of a dynamic lesion, (2) slow and labored, due to difficulty in obtaining a satisfactory respiratory exchange, or (*3*) deep, stertorous, or accelerated The last-mentioned, if unassociated with unconsciousness, is usually caused by other injuries than to the head Terminally, respiration may be slow and gasping Disturbances in respiratory rhythm, such as Cheyne-Stokes respirations, usually indicate a poor prognosis

The hyperpnea in severe head injury may be caused in part by an obstructive collection of secretions in the tracheobronchial system, the result of (*1*) poor respiratory exchange, with pulmonary edema, (2) atony of the bronchial tree, and resultant loss of activity of the ciliated epithelium, (*3*) muscular atony of the smaller bronchioles, possibly due to sympathetic and parasympathetic derangement in the central nervous system

Slow, deep respirations, especially when associated with increasing stupor and a decreasing pulse rate, are a strong indication of a mass lesion — an extradural or a subdural hematoma

Temperature

In moderately severe injury, the temperature rises to 101 to 103 F, then tends to return to normal as improvement sets in In acute injury, the temperature may at first be subnormal or normal, rising gradually several hours to several days after the injury In the presence of other injuries, a subnormal temperature may be part of the shock syndrome, it is then accompanied by a grayish pallor and cold nose, ears, and extremities In severe cerebral contusion, with bloody cerebrospinal fluid, the temperature may be only slightly elevated, and the skin warm and dry, for several hours, then a high fever (104 to 107 F) may develop, together with hyperpnea, rapid pulse rate, and

pulmonary edema. In the absence of unconsciousness, intracranial trauma is in all likelihood not the cause, and other injuries should be looked for

Dehydration, absorption and repair, and infection may all cause a rise in temperature. When fever remains high for more than a few days, search should be made for other causes than the craniocerebral trauma. Pulmonary genitourinary, or meningeal infection may have developed. During convalescence, a sudden rise in temperature, especially after operative procedures may indicate a complicating wound infection

Blood Pressure

By the time a patient reaches the hospital, the blood pressure is at or near normal levels even in patients apparently in shock the blood pressure is well sustained In this respect, the shock of head injury differs from that of other trauma

Occasionally, increasing intracranial pressure, such as caused by a massive hematoma, will lead to a rise in blood pressure,[4, 11] with [illegible] pulse rate and respiration In some cases, high systolic and diastolic pressures may be maintained for several days, then return to normal as improvement sets in Occasionally, the pulse pressure may be as high as the systolic. In our experience, such a pressure [illegible] is associated with a 90 per cent mortality but in children the [illegible] is not nearly so high

On the whole blood pressure data are of little value in [illegible] tracranial pressure, its main value being the evidence [illegible] the general functional competence of the vascular system

Seldom do all the components of the pattern of vital [illegible] in experimental head injury appear in man The pulse [illegible] ration may be slowed, but the blood pressure often does [illegible] expected changes. Nor is is there any definite correl[illegible] between the increase in intracranial pressure and the changes in [illegible] [illegible] (Fig[illegible]

41) This fact must be borne in mind in evaluating the state of a patient with signs of an enlarging intracranial mass For example, a decreased pulse rate and slowed respiration may be associated with focal signs and a deteriorating state of consciousness, or, normal pulse rate and respiration may be associated with an alarming rise in blood pressure; or, a severe bradycardia may be associated with a normal intracranial pressure, clear cerebrospinal fluid, and a normal state of consciousness At times, the cerebrospinal fluid pressure may be normal or subnormal in the presence of an enlarging mass

Shock

In our experience, shock is not a common occurrence in head injury alone, but only when there are other injuries as well In one report on shock in head injury, 46 (6 per cent) of 718 cases manifested a shocklike state in the first 6 hours after injury, and of 119 cases with severe, nonfatal injuries with mild or moderately severe concussion 18 were in a shocklike state [8]

Shock in head injury may be *primary,* caused by activation of the vasomotor centers by the injury, with peripheral vasoconstriction, or *secondary,* caused by blood loss, with resultant decrease in blood volume and peripheral vasoconstriction Pallor in a patient with a moderately severe injury may give the impression of shock, but usually the systolic and diastolic pressures are normal, and the pulse is full, fairly slow, and regular. This is the condition which we have described as primary shock in head injury (*see* Chapter III). Even when the patient appears to be in secondary shock, the pulse may be full and the blood pressure well sustained.

In secondary shock, in addition to the pallor, the skin is cold and moist and the hands and feet are clammy, the pulse is thready, and the blood pressure tends to drop if measures to combat the shock are not instituted. This type of shock is associated with extensive tissue damage and hemorrhage in the head or elsewhere Hypotension persisting for

more than 2 or 3 hours in a patient with a penetrating head injury or with a closed one suggests the presence of profound cerebral trauma or severe blood loss somewhere in the body — the head or elsewhere. In 1 of our cases a continuing hypotension, despite the mildness of the head injury led to the discovery of a ruptured spleen.

In other patients, adrenal insufficiency due to hemorrhages within the substance of the adrenal glands may result in hypotension which resists treatment. Such patients have to be carefully observed and should be treated with cortisone and pressor drugs, such as Levophed bitartrate (levarterenol bitartrate)

Vomiting

This manifestation occurs in about 20 per cent of patients with head injury more frequently in those with milder injuries than in the severely injured in profound injury it is rare It may occur during recovery from unconsciousness.

Nausea and vomiting associated with attacks of vertigo set off by changes in head position may be caused by vagoglossopharyngeal irritation accompanying vestibular trauma. When nausea and vomiting occur with minor head injuries, the cause may be reflex stimulation of the vagoglossopharyngeal system in some cases as a result of swallowed blood being present in the stomach

The vomitus is bloody or the color of coffee grounds in about 15 per cent of the patients The cause may be (*1*) swallowed blood arising from trauma of the oral cavity, (2) an abdominal injury with gastric bleeding and (3) in a few cases, ulceration and hemorrhage of the gastric mucosa due to the effects of a hypothalamic injury

Paralyses and Pareses

Hemiplegia, hemiparesis or paralysis or paresis of some portion of the body may appear soon after injury or may develop only in the course of several hours or days. A contusion or laceration of the motor

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strip in the precentral convolution is more apt to cause an immediate paralysis or paresis A slowly progressive paralysis, on the other hand, usually indicates a contralateral dynamic mass lesion which is compressing the motor cortex, occasionally, however, the paralysis or paresis and the lesion may be on the same side In the latter cases, the contralateral motor area may be contused and edematous, or, more commonly, the cause may be uncal herniation and compression of the brain stem contralateral to the mass lesion The improvement in traumatic hemiplegias and hemipareses, as well as in aphasia, is fairly complete, in contrast to the outcome in cerebrovascular diseases and brain tumors

In an occasional patient with fairly complete hemiplegia or hemiparesis, there is no history of unconsciousness after the head injury. In such cases, the possibility of a complicating cerebrovascular accident or of a traumatic thrombosis of the internal carotid artery in the neck or of the middle cerebral artery at its junction with the internal carotid artery should be considered.

More serious paralyses — triplegia and quadriplegia — are usually due to. (*1*) involvement of the brain stem centers, (*2*) trauma of the sagittal sinus, with thrombosis, and (*3*) diffuse cerebral injury, with involvement of both hemispheres Holmes and Sargent [20] first described the symptomatology of superior longitudinal sinus trauma paresis of the lower limbs and of one or both upper limbs Choreiform movements may occur in patients with brain stem involvement, this is more commonly seen in the younger age groups, possibly because they more frequently survive brain stem injury than do adults When both pyramidal tracts are involved (usually in diffuse cerebral injury), there may be pseudobulbar manifestations, including nasal speech, and an inability to swallow in the early stages of the trauma Swallowing usually improves rapidly, but the nasal, possibly somewhat slowed, speech may remain for years (Fig 42)

Brain stem syndromes rarely occur Occasionally, there may be Weber's syndrome (ipsilateral third nerve paralysis and contralateral paresis of the lower face, tongue, and extremities) It is worth em-

phasizing that this syndrome may also be caused by involvement of the third nerve in an uncal herniation on one side and compression of the motor strip over the hemisphere by a hematoma, producing a contralateral hemiparesis or hemiplegia. Benedikt's syndrome (ipsilateral third nerve paralysis, with contralateral hemianesthesia or hemihypesthesia, and evidence of red nucleus involvement consisting of tremors in the hemihypesthetic side) we have seen in 2 cases only. These tremors, supposedly more severe at the end of a movement, are usually called terminal tremors.

Various combinations of neurologic deficits may point to a deep-seated lesion of the internal capsule on one or the other side. For example, a homonymous hemianopsia associated with a paresis or paralysis suggests subcortical involvement.

Catatonic states almost always result from a left frontoparietotemporal injury, and aphasia is an associated phenomenon.[18, 24, 30] The catatonic patient may be conscious or semiconscious; a limb placed in a certain position remains until fatigue causes it to drop.

Sensory Abnormalities

Sensory disturbances of cortical origin are uncommon in head injury with the exception of parietal lobe trauma. Even in such cases, sensation is often normal except for a contralateral astereognosis in the hand. Some loss of contralateral tactile and vibratory senses may occur with injury of the postcentral gyrus. In penetrating brain injuries, with trauma of the cortex over the parietal lobe, Head[37] found loss of two-point discrimination and position, while pain and touch sensations were not impaired. Marshall,[43] on the other hand, found this to be true only in cases with extensive injury of the parietal area; in such cases, the thalamic centers and the contralateral cortex took over the recognition of pain and touch. Smaller and limited cortical lesions, however, resulted in impairment of superficial and deep pain sensation, as well as of two-point discrimination and position senses.

Speech Defects

Disturbances of speech result from involvement of the left postero-inferior frontal convolution (Brodmann's area 44, Broca's convolution) in right-handed individuals, or from involvement of the postero-superior temporal gyrus With injury of the former, there is usually a motor or expressive type of aphasia, with injuries of the latter, a sensory aphasia Anomia, the inability to name objects, is frequently seen in patients with temporal lobe lesions A depressed skull fracture in the left frontoparietotemporal area may cause a motor aphasia, and sometimes jargon speech An occasional patient is unable to understand spoken language As a patient becomes more alert and cooperative, signs of sensory aphasia, alexia, agraphia, and apraxia may appear.

The prognosis for eventual return of function is relatively good The aphasia usually improves despite the large amounts of brain which might have been sacrificed during debridement

Convulsive Seizures and Decerebrate Rigidity

Convulsive Seizures

The incidence of seizures — whether focal (jacksonian) or generalized (grand mal) — in the immediate period after an acute head injury is small, probably well below 10 per cent Their occurrence in this phase of head injury is of little, if any, prognostic significance with regard to the possibility of posttraumatic epilepsy.

In our 1952-1954 series, 72 patients had one or more convulsive seizures (20 jacksonian, 52 generalized), and 42 per cent of them died An analysis of these figures in relation to the age of the patient revealed that, on a percentage basis, seizures were much more common in the group under the age of 10 than in the patients between the ages of 11 and 50

Seizures may be caused by a generalized increase in intracranial pres-

sure, subarachnoid or intraventricular hemorrhage, cerebral contusions and lacerations, extradural or subdural hematoma, cerebritis, meningitis, and cerebral abscess. A focal or a generalized seizure may be the first sign of a posttraumatic complication meningitis, cerebritis, or abscess.

Focal seizures suggest the presence of a discrete cerebral lesion commonly an extradural or subdural hematoma compressing some area of the motor strip, or contusion of the motor cortex. Unilateral or bilateral facial and circumoral seizures may occur with contusions and lacerations of the frontotemporal junction [25] If treatment is delayed, or the hematoma is not evacuated, the focal seizures may become generalized

The grand mal seizure occurs less frequently than the jacksonian one, it is a more frequent complication of head injury in infants and children than in adults It may occur soon after injury, as the patient is recovering consciousness, or later, or even after the evacuation of a subdural hematoma. Occasionally, the seizures will follow an attack of petit mal or of decerebrate rigidity Progress to status epilepticus is rare, but has been known to occur

The electroencephalogram may show diffuse or focal abnormalities, but these tend to disappear as the acute phase of the injury is passed and improvement sets in. However, some focal abnormalities may persist. Generalized spike-and wave patterns in the acute phase, in the absence of diffuse or focal abnormalities, probably antedate the injury

Decerebrate Rigidity (Fig 42)

This, too, is uncommon in the acute phase of severe head injury It may be caused by (*1*) trauma of the brain stem, (2) herniation of the brain stem or the temporal lobe, or of both, as a result of cerebral edema or of space-taking supratentorial lesions [12] (*3*) large extradural or subdural hematoma or cerebrospinal fluid collection, (*4*) bilateral cortical contusions of the motor area and of Brodmann's area 8 The decerebrate attitude is characterized by extensor rigidity, somewhat retracted head, adducted and internally rotated upper extremities, ex

tended and hyperpronated forearms, flexed hands, and fingers usually flexed at the metacarpophalangeal joints but extended and adducted distally Bilateral signs of pyramidal tract involvement and Babinski's signs are present The latter may be continuous, particularly in the younger age groups in whom opisthotonos and nuchal rigidity are also more frequent The jaw is fixed, and in some there is increased salivation and foaming at the mouth An attack of rigidity lasts a few minutes to several hours Short attacks are apt to recur

The rigidity may be unilateral, changing from one side to the other, but generally the patient becomes completely rigid on being moved or stimulated In almost every case of decerebrate rigidity, the cerebrospinal fluid is bloody, and the pressure is usually elevated

The rigidity becomes less pronounced as the patient's state deteriorates Most adults die, but many children recover, particularly those in whom the rigidity was caused by a mass lesion which could be evacuated

Headache

Headache or pain of the back of the head and neck is the most common symptom of head injury It may be a major complaint in mild injury, and may be absent in severe injury The headache in severe injury may become apparent as the patient emerges from the unconscious state, in patients who have not been unconscious initially, the

Fig 42 Paralytic states with bulbar and pseudobulbar phenomena (*a*) Patient unconscious for 7 weeks after injury, lowered mentation for 4 months, with little improvement, after initial period of generalized rigidity, with sustained Babinski's signs and extended and internally rotated extremities, patient photographed at stage of crossed paralysis with a sustained Babinski's sign on left and paresis of right upper limb with extension and internal rotation of upper extremity and eversion of hand and flexion of fingers, patient improved until he was able to move about satisfactorily, small brain stem hemorrhages were undoubtedly present (*b*) Patient unconscious and mute until death, 3½ months after injury, bilateral rigidity and extensor spasms of upper and lower extremities, with greater involvement of left side, continuous bilateral Babinski's signs, autopsy revealed extensive bilateral cerebral injury, particularly over frontoparietotemporal area, and some brain stem involvement.



onset of headache may signal an increase in intracranial pressure

The following factors may play a role in posttraumatic headache: (*1*) changes in vascular tone and caliber, for example, dilatation or constriction of the blood vessels at the base or of the extracerebral vessels,[14] (2) trauma of the dural blood vessels, (*3*) trauma of the hypothalamus, causing disturbances of vascular tone, (*4*) blood in the subarachnoid space, resulting in altered cerebrospinal fluid dynamics and traction on the larger vessels at the base and over the hemispheral convexities, (5) increase in intracranial pressure from hemorrhage, hematomas, or edema; (*6*) in the absence of increased intracranial pressure, traction on the larger vessels at the circle of Willis when head position is changed; (7) associated injury of the muscles, ligaments, and nerves in the neck (whiplash injury), a particularly important cause of prolonged posttraumatic headache

The headache is usually generalized, but occasionally it is limited to one side of the head or to the area of scalp injury [30] The generalized headache after injury at the craniospinal junction, such as the whiplash injury, is explained by the extensive distribution in the scalp of sensory fibers from the upper cervical region and from the cervical plexus [11, 31] A general hypotension, with traction on the cranial and intracranial blood vessels on changes in position, may be the basis of the headaches which some patients have on getting up, and which disappear when a recumbent or semi-Fowler position is resumed Fatigue, noise, excessive heat or light, straining, lifting, or bending may initiate or aggravate the posttraumatic headache, and leisure and freedom from tension relieve it.

Migraine or periodic headaches antedating the head injury may become more severe after injury, and a migraine quiescent for years may again become active after the injury Whether a neurosis or justification syndrome may be the cause in such cases is not clear, and a fact hard to establish, but the injury might possibly be a precipitating factor Psychic trauma, such as divorce or financial difficulties, are said to have the same effect

The headaches usually improve in the course of some weeks or months. In patients who complain of both dizziness and headache, the dizziness usually subsides first. Complaints of headache which last for a longer time may be based on compensation considerations, with financial benefits continuing so long as the complaint is validated

Giddiness, Dizziness and Vertigo

Giddiness or dizziness is a sensation of unsteadiness, the two differing only in degree, but vertigo is a sensation of true rotation Vertigo, including postural vertigo, is caused by a derangement of the vestibular end organ, dizziness and giddiness, by a derangement of the primary vestibular nuclei and central vestibular pathways in the brain stem.[12, 13]

Giddiness or dizziness is not infrequent in head injury, of 100 convalescent patients studied for cochleovestibular dysfunctions after head injury, 50 complained of dizziness. Giddiness is common in milder grades of head injury, and is particularly apt to occur with changes in body position, as in rising from a recumbent position or lying down after standing Patients soon learn to move with deliberation in order to prevent attacks. Vestibular function is not impaired in these cases, but the reaction to stimuli may be increased As a rule, giddiness disappears within several weeks to a few months. When a patient with a minor head injury complains of headaches and giddiness beyond the convalescent stage, psychoneurotic factors may be playing a role.[13]

An attack of a true, turning vertigo is of short duration It may be associated with a spontaneous nystagmus, with the quick component to the opposite side, disturbed balance, past pointing and autonomic disturbances, such as nausea vomiting perspiration, a cold clammy skin rapid pulse, and lowered blood pressure. The response to caloric testing in such a case may be assymetric. Postural vertigo with errors of sensation, such as tilting of the bed, rising of the floor, or falling of the ceiling a feeling of uncertainty or of drunkenness, may be due to a peripheral vestibular injury in which case the patient s caloric responses

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Giddiness or dizziness is not infrequent in head injury of 100 convalescent patients studied for cochleovestibular dysfunctions after head injury, 50 complained of dizziness. Giddiness is common in milder grades of head injury and is particularly apt to occur with changes in body position as in rising from a recumbent position or lying down after standing Patients soon learn to move with deliberation in order to prevent attacks. Vestibular function is not impaired in these cases, but the reaction to stimuli may be increased. As a rule, giddiness disappears within several weeks to a few months When a patient with a minor head injury complains of headaches and giddiness beyond the convalescent stage, *psychoneurotic factors may be playing a role.*[13]

An attack of a true, turning vertigo is of short duration It may be associated with a spontaneous nystagmus with the quick component to the opposite side disturbed balance, past pointing and autonomic disturbances such as nausea, vomiting perspiration a cold clammy skin rapid pulse, and lowered blood pressure The response to caloric testing in such a case may be assymetric. Postural vertigo, with errors of sensation such as tilting of the bed rising of the floor or falling of the ceiling a feeling of uncertainty or of drunkenness, may be due to a peripheral vestibular injury, in which case the patient s caloric responses

are abnormal,[21] or to a derangement in the utricular macula. The latter may result from transmission to the vestibule of the pressure pulse set up by the trauma which displaces the otolithic membrane in the utricular macula; acceleration and gravity may have the same effect.

These types of vertigo usually subside within several weeks, since the vestibular nuclei eventually compensate for the unbalanced impulses which the deranged labyrinth may initiate.

The usual disequilibrium in patients with head injury, however, is more suggestive of brain stem lesions, with involvement not only of the vestibular fibers but also of the tectospinal, rubrospinal, and spinocerebellar pathways. Leidler[30] gives the following symptoms and signs as strongly indicative of a central nervous system lesion: (*1*) normal hearing, combined with ataxic vertigo, or with hyperexcitability of one labyrinth, or with spontaneous nystagmus; (*2*) headache and vertigo; (*3*) positional nystagmus; (*4*) spontaneous nystagmus without vertigo; (*5*) preponderance of the slow phase of the nystagmus; (*6*) rhythmic disturbances in vestibular reaction. Another set of signs and symptoms are listed by Van Egmond and associates[77] as indication of the central nervous system origin of dizziness or vertigo: (*1*) spontaneous nystagmus toward a dead labyrinth, or undiminished spontaneous nystagmus for 1 week; (*2*) long-lasting rotational nystagmus; (*3*) vertical nystagmus; (*4*) normal turning reactions without caloric excitability; (*5*) monophasic reactions without a quick phase; (*6*) somnolence when head position is changed.

Positional nystagmus is a spontaneous nystagmus which appears when the head assumes particular positions in space.[16] Ocular movements are usually normal in patients who have residual partial or complete deafness after head injury; on the other hand, the occurrence of positional nystagmus in patients whose hearing is unimpaired is further indication that a brain stem lesion may be the cause of the nystagmus.

Of our 100 convalescent patients mentioned earlier, 23 had spontaneous nystagmus, and 10 positional nystagmus.

Cupulometry, a turning test with small regulable stimuli such as are provided by the ordinary movements of life, has been suggested as a means for obtaining quantitative data on vestibular function with respect to nystagmus and cessation of vertigo[22]. The duration of nystagmus and vertigo after a subject has been rotated at specified speeds, plotted on a logarithmic scale, gives a graph called the cupulogram. Cupulometry might be an objective test of the subjective complaints of patients with vestibular disturbances after head injury.

In the acute stage of total unilateral loss of vestibular function, such as occurs in transverse fractures of the petrous bone, the nystagmus and vertigo may be severe. The horizontal nystagmus is toward the normal side, the subjective vertigo is in the same direction as the nystagmus, and the past pointing is in the opposite direction. These symptoms subside within a month of destruction of the end organ, and in the chronic stage spontaneous vestibular symptoms are absent. Nevertheless, patients with a dead labyrinth may still have considerable disability despite eventual compensation by the remaining labyrinth. These patients suffer from errors in the sense of location and space when they are in the dark, and sudden changes in position may cause an attack of vertigo.

Symptoms and Signs Referable to Ear

Bleeding

Minor or extensive bleeding from one or both ears is fairly frequent in head injury, particularly with skull fracture, in about 30 per cent of some reported series[23, 24] and in 24 per cent of a recent series of ours. In this series, 164 of the 185 cases with bloody otorrhea had skull fractures. The mortality rate in an older series was 31 per cent of cases with unilateral bleeding and 66 per cent with bilateral bleeding[16, 21]. The mortality rate in our recent series is considerably lower—17.5 per cent—probably as a result of the use of antibiotics and sulfonamides, and of earlier and frequent otoscopic examinations.

The bleeding may be simply the result of a laceration within the external auditory canal or of rupture of the tympanic membrane, in such cases, the bleeding is minor. A profuse hemorrhage indicates injury of the transverse or superior petrosal sinuses, the jugular bulb, the tympanic venous and arterial plexuses, or of an injured artery, *e.g.*, the middle meningeal Involvement of the carotid artery in a temporal bone fracture will cause profuse bleeding not only from the ear but also from the nose and mouth. Delayed (up to 1 month after injury) or late bleeding from one ear may be due to involvement of a small vessel in an osteomyelitic area or to osteitis with erosion [20]

Even in the absence of a history of bleeding from the ear after head injury, blood clots, ecchymoses, or a hematoma on the walls of the external auditory canal, most often on the posterior and superior walls, may be found on examination An ecchymosis over the mastoid area, Battle's sign, 4 or 5 days after injury, is usually a sign of temporal bone fracture. Another, although rare, sign of transverse fracture of the temporal bone is hematotympanum, a blue discoloration of the drum and a bulging of the tympanic membrane

Cerebrospinal Fluid Otorrhea

The reported incidence varies, depending on the investigator and the series, from 2 to 20 per cent.[4, 22, 24, 61] It may be difficult to identify the otorrhea because there is bleeding from the ear or because the otorrhea is so scanty and of such short duration that it is not noticed On the other hand, it may be copious and persist for several weeks Onset of the otorrhea may be delayed,[5, 22] the presence of cerebrospinal fluid behind the tympanic membrane may be recognized by the appearance of a definite fluid level in the tympanic cavity [58] In rare cases, macerated cerebral tissue is discharged along with the fluid

Hearing Abnormalities

Head injury frequently affects hearing, most usually causing loss of hearing acuity but occasi[illegible]easing the acuity. Loss of hearing

may be temporary or permanent, partial or complete, unilateral or bilateral, a conduction or a nerve defect, and become apparent immediately after the injury or after some time.

In Grove's[22] series, of 108 cases of longitudinal temporal bone fractures, 79.5 per cent were deaf on the ipsilateral side, and 54.6 on the contralateral side. In our series of 25 cases of such fracture, there were 9 of nerve deafness and 13 of conduction and nerve deafness. In transverse fractures of the temporal bone Grove reported complete deafness on the homolateral side in all of a series of cases, and considerable loss on the contralateral side in 65 per cent. Loss of cochlear function without impairment of vestibular function has been reported in cases with fracture lines extending through the pyramid demonstrated on the roentgenogram.[37, 47]

Hearing loss is less frequent with fractures not involving the temporal bone in a series of Grove's[22] of 49 cases, some nerve deafness was found in 45 *ears* out of the total 98, in 13 of our cases with skull fracture, there were 8 of nerve deafness (6 bilateral 2 unilateral), and in 46 without skull fracture varying degrees of nerve deafness in 28 (mild in 12)

The bone or conduction type of deafness is usually due to temporal bone fracture, and in such cases there may be bleeding from the ear and a torn tympanic membrane.

Early audiometric studies may show uniform loss of hearing for all frequencies. Later, the loss due to lesions of the conductive mechanism disappears whereas perceptive loss remains almost unchanged, if changed at all.

Tinnitus

Between 30 and 40 per cent of patients with cochleovestibular disturbances after head injury complain of tinnitus. It is almost always caused by injury to the end organ in the petrous bone Usually the tinnitus is confined to the involved ear

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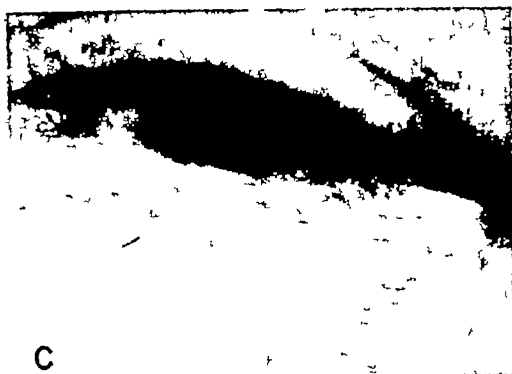










Fig 44. Ocular manifestations in head injury. (*a*) Left hematoma, due to comminution of orbital floor (*b*) Right periorbital hematoma (*c*) Prolapse of orbital fat. (*d*) Retrobulbar hemorrhage and proptosis of left eye (d^1) and appearance after recovery without surgical intervention (d^2) (*e*) Right enophthalmos (e^1), due to comminution of orbital roof and supraorbital ridge, and appearance after repair (e^2).

As a rule, conservative measures may be used safely in such patients. In others, the pupils are dilated and unresponsive to light, and the corneal reflexes lost; these are the patients with probable irreversible cerebral damage who may die within hours after injury. Occasionally, there is dissociation between the pupillary reflex to light and the corneal reflex to touch; thus, in the first 24 hours the pupillary reflex may be absent while the corneal reflex is elicited. In such cases, recovery is possible.

Bilateral pupillary constriction with almost complete or complete loss of reaction to light does not always indicate major cerebral damage. Unilaterally dilated, fixed, or sluggishly reacting pupils are usually caused by mass lesions, cortical damage, partial or complete paralysis of the oculomotor nerve (frequently due to uncal herniation), or severe optic nerve injury.

Loss of the corneal reflex results from injury of the trigeminal or facial nerves. Bilateral loss of the reflex is a grave prognostic sign, whereas unilateral loss is an indication that there is no irreversible brain stem trauma. An Argyll Robertson pupil after head injury may be due to involvement of the ciliary ganglion in the orbit or to selective damage of the pupillary constrictor fibers, particularly if vision is not affected.[48]

In our large 1952-1954 series there were 143 patients with pupillary abnormalities. In 96 patients the pupils were unequal; the mortality rate in this group was about 44 per cent. In 30 patients both pupils were dilated but reacted to light; the mortality rate in this group was 56 per cent. In 16 patients the pupils were dilated and fixed; 15 of this group died — a mortality rate of about 94 per cent.

Exophthalmos and Enophthalmos (Figs. 43-44)

Exophthalmos may occur with (1) depressed fractures which compress the contents of the orbit, (2) penetrating fractures involving the orbit, (3) intra- or periorbital hemorrhage or edema, or (4) carotid artery–cavernous sinus fistula. Severe intraorbital hemorrhage, as in

arteriovenous fistula (*see* Fig 49), may cause a progressive exophthalmos

A mild enophthalmos is rather common in head injury, and usually results from a fracture involving the roof or floor of the orbit, with herniation of the orbital contents Comminution of the roof or floor may tear Tenon's capsule, permitting herniation of the orbital fat Rarely, enophthalmos may result from injury of the sympathetic pathways

Fundus

Soon after an acute head injury, funduscopic examination may fail to reveal the extent of cerebral trauma Hemorrhage may occasionally be detected, particularly retinal hemorrhage in infants, children, and the younger age groups, the latter indicates direct retinal injury by the initial impact Retinal edema may be apparent within the first 10 to 24 hours, if there is a rapidly expanding intracranial hemorrhage, a pronounced papilledema, with venous dilatation and retinal hemorrhages, may occur within 12 to 20 hours Papilledema 4 to 6 days after injury is usually an indication of extradural, subdural, or intracerebral hematoma, but occasionally there may be no mass lesion, in such cases, the results of pneumoencephalography or ventriculography may be normal Papilledema, retinal hemorrhages, and venous engorgement with arterial blood, in addition to exophthalmos, may develop within 48 hours in a patient with a carotid artery–cavernous sinus fistula Tearing of the optic nerve in orbital fractures with evulsion may cause a depressed disk

In sudden blindness due to optic nerve injury, the fundus is usually normal at first, although the retina may be edematous in some cases; in the course of 6 to 8 weeks, the optic disk grows progressively pale as a result of primary atrophy (Fig 45)

Visual Fields

Defects in the visual fields occur more commonly with open than with closed head injuries; in the latter, they are usually asymmetric

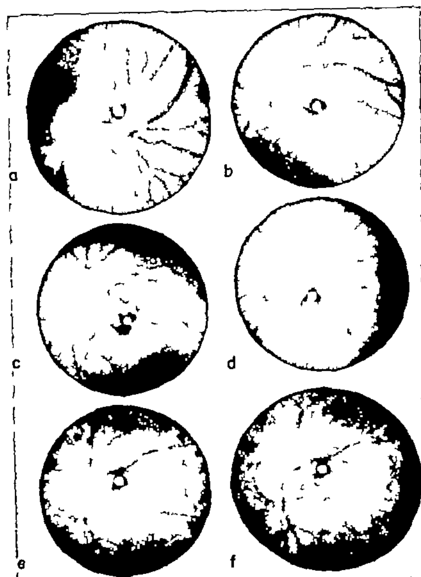


Fig 45 Optic *atrophy in head injury results of funduscopic* examination of patient blind in one eye, 1 day to 5 months after injury Atrophy is usually noted after 21 days (*b*) 7 days, (*c*) 14 days, (*d*) 24 days, (*e*) 60 days, and (*f*) 5 months after injury

Constriction *of the visual fields* is common soon after a head injury

A horizontal hemianopsia is attributed by Turner[34] to a compromised blood supply of the optic nerve as it passes through the optic foramen. A ring scotoma has been reported as being caused by callus formation at the optic chiasm.[41]

A homonymous hemianopsia or *quadrantanopsia* may occur with

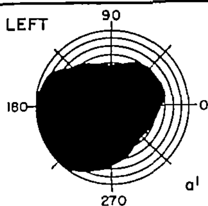
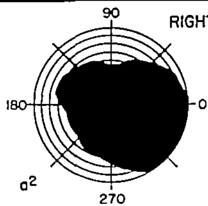
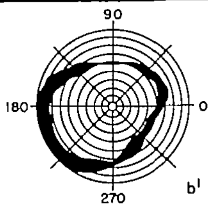
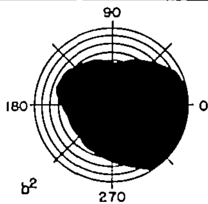
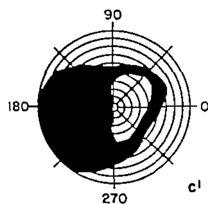
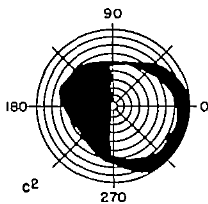
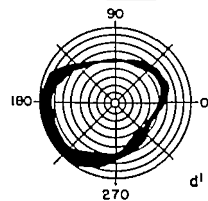
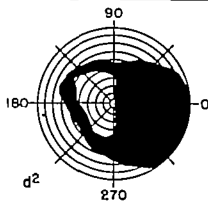
open injuries in the temporal area, in 1 of our patients, a temporal hemianopsia was probably due to injury of the medial fibers of the optic nerve, anterior to the chiasm Quadrantanopsias may also occur with injuries of the parieto-occipital region and of the occipital tip (Fig 46) Spalding[54] has described paracentral scotomas with cortical injuries in the occipital area, although Holmes[30] apparently did not find them in patients from World War I Occasionally, compression of the posterior cerebral artery by uncal herniation may be the cause of a homonymous hemianopsia [44] A lower quadrantanopsia results from injury of the upper portion of the anterior component of the optic radiation, an upper quadrantanopsia from injury of the lower portion of the radiation Sector defects result from involvement of the intermediate fibers of the radiation Defects in peripheral vision are due to injury of the calcarine cortex anteriorly, defects of central vision, to injury of the calcarine cortex posteriorly The depth of the calcarine fissure contains cells subserving the horizontal meridian of the visual fields

Injury of the posterior aspect of the head midoccipitally may cause a temporary blindness, which disappears after several hours. The cause may be a temporary bilateral involvement of the calcarine centers in Brodmann's area 17 by contusion and edema Bilateral blindness or homonymous hemianopsia may occur with injury at the confluence of sinuses

Blindness (Fig. 45)

A frequent cause of blindness is optic nerve involvement in the orbit or in the optic foramen The head injury causing the blindness may be minor or severe, closed or open, and the blindness may be unilateral or bilateral, and permanent or temporary Both direct and indirect injuries to the globe, with hemorrhage into the aqueous and vitreous

Fig 46 Visual field defects in acute head injury (*a*) Complete blindness after right temporoparietal skull fracture (*b*) Blind right eye after closed head injury, result of optic nerve inspection essentially negative (*c*) Left homonymous hemianopsia after injury of right occipital lobe (*d*) Right temporal hemianopsia, optic nerve normal on inspection


LEFT
90
180
0
270
a1

90
RIGHT
180
0
270
a2

90
180
0
270
b1

90
180
0
270
b2

90
180
0
270
c1

90
180
0
270
c2

90
180
0
270
d1

90
180
0
270
d2

humors and the retina, may cause partial or complete blindness. Penetrating frontal fractures are a particularly frequent cause of vitreous and retinal hemorrhages, with resultant blindness Retinal separation occurs occasionally

Ophthalmoplegias (Fig. 47)

Ocular and extraocular palsies may be caused by. (*1*) involvement of extraocular muscles and nerves and of the eyeball in orbital injuries, and (2) involvement of the nerves subserving extraocular movements and pupillary size in their course from the brain stem to the orbit Any or all of the extraocular nerves — the oculomotor, the trochlear, and the abducens — may be injured when increased intracranial pressure causes uncal herniation and brain stem dislocation, or when the bone structures about the cavernous sinus are injured, or when there is trauma around the supraorbital fissure In our series of 1,285 cases there were 6 of oculomotor and 5 of abducens nerve paralysis, in Turner's[16] series of 1,550 cases, 15 of oculomotor and 15 of abducens nerve paralysis

Complete oculomotor paralysis is manifested by. (*1*) ptosis, (2) dilatation of the pupil, with loss of accommodation and of direct and consensual light reflexes, (*3*) inability to move the eyeball inward, upward, and downward, and (*4*) mild exophthalmos Partial oculomotor injury is shown by partial ptosis, somewhat dilated pupil, diplopia, but no gross impairment of ocular movements Weber's syndrome (oculomotor paralysis on the side of the lesion and a contralateral hemiplegia) occurs when the third nerve nucleus and the pyramidal tract in the brain stem are injured Complete or partial oculomotor paralysis in a conscious patient whose state is improving is an indication that the paralysis is not caused by a mass lesion, whereas in a patient whose state of consciousness is deteriorating it is likely to be caused by an extradural, a subdural, or an intracerebral hematoma, and associated uncal herniation. The herniation may also cause partial paralysis with pupillary dilatation. Bilateral oculomotor nerve involvement may occur with

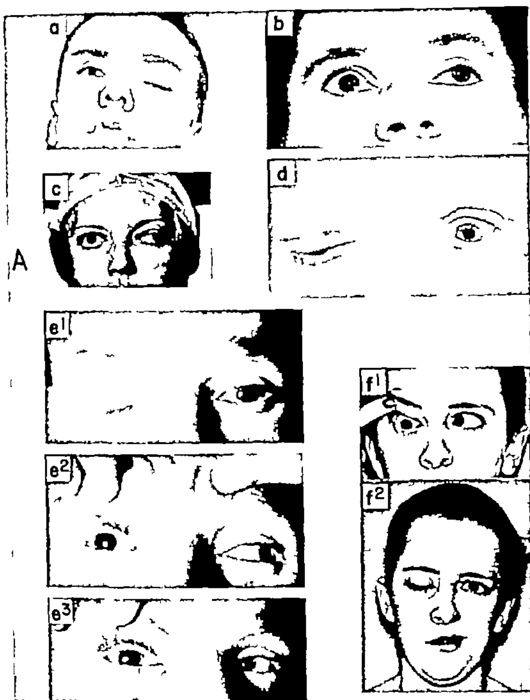


Fig. 47 Extraocular paralyses in head injury (*A*) Left oculomotor nerve paralysis (*a*) (*b–c*) Left oculomotor nerve paralysis, with partial recovery (*d*) Left oculomotor nerve paralysis, with partial recovery: patient, looking downward with right eye, is unable to rotate left eyeball downward or lower left eyelid. (*e*) Complete right ophthalmoplegia, due to basilar fracture. (*f*) Complete right ophthalmoplegia, with peripheral right facial nerve paralysis, due to fracture of petrous bone. (*Continued on next page*)

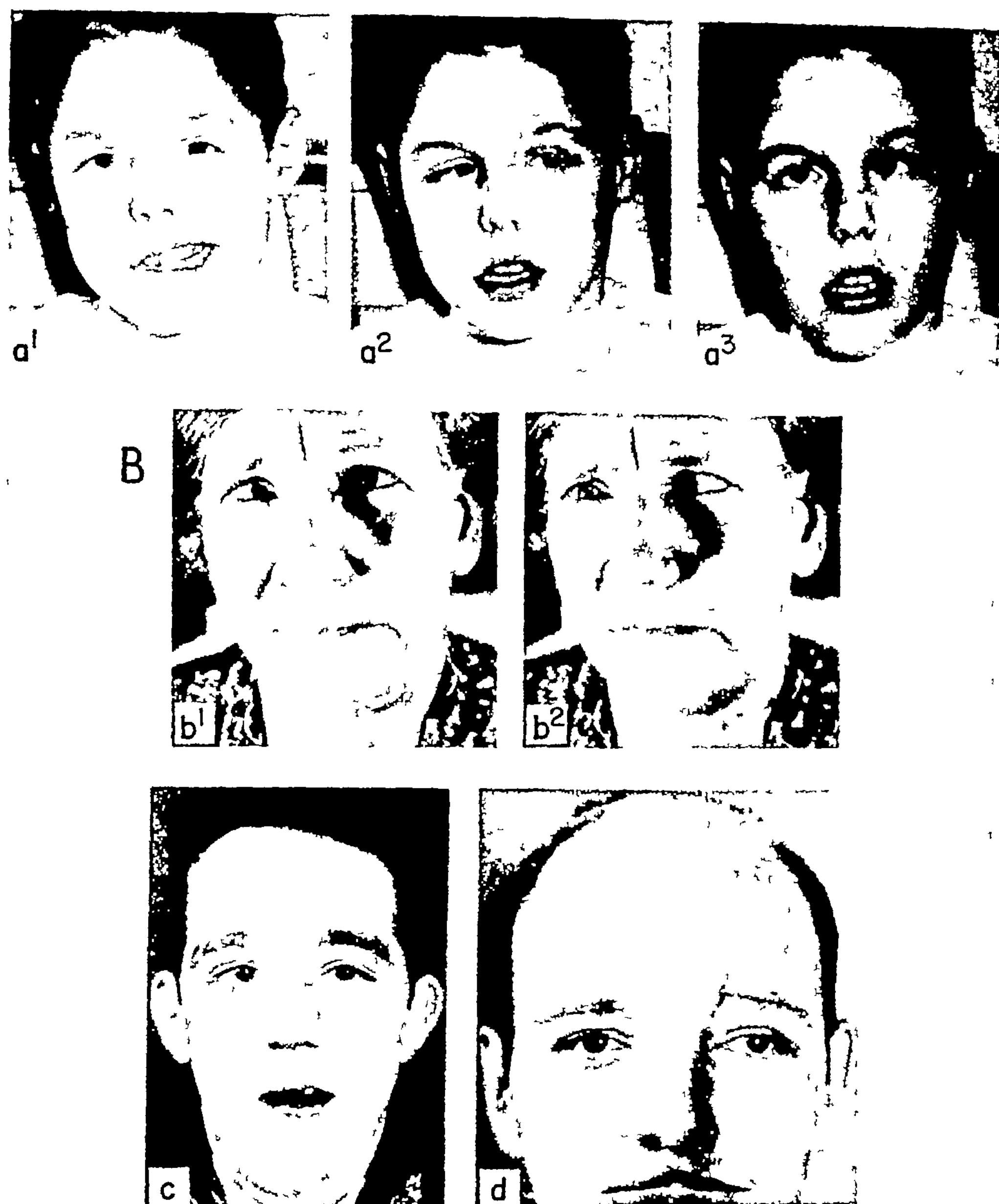


Fig 47 (*continued*). (*B*) Bilateral abducens nerve paralysis and peripheral right facial nerve paralysis (*a*) (*b*) Bilateral abducens nerve paralysis (*c*) Right abducens and peripheral facial nerve paralysis (*d*) Left eye blind with slight outward rotation, due to optic nerve injury

unilateral or bilateral extradural or subdural hematoma, evacuation of the hematoma leads to rapid improvement.

The trochlear nerve mediates, in part the medial upward and downward movements of the eye. Isolated trochlear nerve paralysis is rare in head injury, but the superior oblique muscle may be affected in depressed fractures involving the medial aspect of the orbital wall. Diagnosis of trochlear nerve involvement by routine neurologic examination is difficult.

Partial or complete abducens nerve paralysis, or inability to move the eyeball outward, may occur with head injuries of varying severity. Occasionally, the paralysis is bilateral and sometimes is associated with unilateral or bilateral facial nerve palsy, indicating a nuclear lesion at the genu of the facial nerve. Abducens nerve paralysis is often of no lateralizing value in the presence of a mass lesion. Improvement is usually rapid, but if the condition has not improved by the end of 3 months, some permanent paralysis may be expected.

Paralysis of ocular movements may occur in head injuries complicated by a carotid artery–cavernous sinus fistula. The lateral rectus muscle is involved frequently, and eventually all of the extraocular muscles are affected.

Paralysis of upward and downward gaze indicates brain stem damage at or about the superior colliculi. Such signs are uncommon in head injury, since involvement of these regions is usually fatal.

Conjugate Deviation

The eyes may deviate to one side or the other in severe head injury with cortical contusion and sometimes in the presence of a hematoma. A destructive lesion in the frontal eye fields will cause homolateral deviation; irritative lesions and brain stem lesions contralateral deviation.

Fixation

Inability to fix the eye upon an object may be due to a lesion of the occipital lobe and the visual cortex, or to an involvement of the superior longitudinal fasciculus in the brain stem

Diplopia

Double vision and blurred vision are common complaints during convalescence from head injury The causes of diplopia are: (*1*) displacement of the eyeball in fractures of the orbital roof or floor, (*2*) involvement of extraocular nerves or muscles, (*3*) intraorbital involvement of muscular and neural structures associated with local muscle and nerve injury.

Visual Agnosia

One of the major deficits in patients who survive profound head injury is a visual agnosia and an alexia (inability to recognize words) based on the visual agnosia An inability to recognize the opposite half of the body may occur with lesions of the forward part of the left occipitoparietal cortex in right-handed individuals[17, 49]

Symptoms and Signs Referable to Nose

Deceleration injuries, particularly in automobile accidents, may be associated with facial involvement and injury to the nose and paranasal sinuses. Bleeding from the nose, cerebrospinal fluid rhinorrhea, and olfactory abnormalities are common

Nasal Bleeding

It may be minimal, or so profuse as to necessitate major efforts for its arrest. Occasionally, the bleeding may be due to a tear of the carotid artery at the base of the skull, with hemorrhage into the middle ear and, via the eustachian tube, into the nose In other instances, local injury of the turbinates and septum may be the cause of bleeding so

profuse that it can be stopped only by nasal packing Bleeding associated with cerebrospinal fluid leakage is common soon after injury Later, the rhinorrhea may continue, the cerebrospinal fluid becoming clear and readily identifiable. Bleeding from the nose is seen in 25 per cent of patients with head injury

Cerebrospinal Fluid Rhinorrhea

This is a rather rare sequel of head injury It may occur in 1 to 3 per cent of patients. Traumatic cerebrospinal fluid rhinorrhea may also follow surgical removal of a nasal polyp which may actually be an evagination of the subarachnoid space and dura through an opening in the roof of the nasal cavity In many cases, bed rest suffices for spontaneous recovery, but in some the rhinorrhea stops only temporarily, later to be followed by recurrences

The diagnostic features of cerebrospinal fluid rhinorrhea include (*1*) The fluid contains glucose, nasal secretions do not. (*2*) The fluid is clear, and the discharge is intermittent. (*3*) The protein content of the fluid is low, that of ordinary nasal secretions high, a handkerchief moistened with fluid does not stiffen on drying (*4*) Drainage can usually be increased by placing the patient with his head between his thighs in a flexed position. (*5*) The fluid usually drains from one nostril rather than from both. (*6*) Anosmia is frequently associated with the rhinorrhea. A dye such as fluorescein injected into the spine may be recovered in the nasal discharge in 30 to 90 minutes, as noted by Constantin[6a] and by Fox[11a] among others

In some patients there is little or no discharge of fluid despite the presense of a patent craniomasal fistula. These patients may have repeated attacks of meningitis. In a patient with a head injury recurring meningitis should therefore arouse the suspicion of a patent craniomasal fistula.

Table 5 summarizes the findings in 22 cases of our series. It is noteworthy that in 2 of the patients the fistula was closed only on the second attempt.

TABLE 5 *Data on 22 Cases of Traumatic Cerebrospinal Fluid Rhinorrhea*

Data	Number of cases		
	Total	*Recovered*	*Fatal*
Acute	12	11	1
Recurrent	10	7	3
Complications			
In acute Pneumocephalus	3	3	0
Meningitis	1	0	1
In recurrent Brain abscess	2	1	1
Pneumocephalus	1	1	0
Meningitis	2	0	2
Treatment			
In acute Surgical	6	6	0
Conservative	6	5	1
In recurrent Surgical	5	5	0
Conservative	5	2	3

Olfactory Nerve (Cranial I) Involvement

Injury of the olfactory nerve causes a loss of smell (anosmia), or a perversion of the sense of smell (parosmia) According to Lewin,[40] the sense of smell is affected in about 5 per cent of patients with head injury We have found the incidence to be even lower (about 3 per cent), except in cases complicated by cerebrospinal fluid rhinorrhea, in which case 4 out of 10 patients lose the sense of smell, the incidence rises to 80 per cent if the cranionasal fistula is repaired

The nerve may be injured in anterior fossa fractures in the neighborhood of the cribriform plate, or in deceleration injuries of the forehead or the occipital area which may result in a tear of the nerve connections in the cribriform plate area

Many patients recover some function within 2 years of injury In this period, parosmia may become evident, and the patient is unable

to recognize test objects. In our experience, anosmia does not materially affect the patient's sense of taste, at least for basic flavors

Symptoms and Signs Referable to Cranial Nerve Injuries

Results of injuries of the first, second, third, fourth, and sixth cranial nerves have just been discussed The other cranial nerves which may be involved in head injury are principally the trigeminal and the facial. The glossopharyngeal, vagus, accessory, and hypoglossal nerves are seldom involved in the usual, closed type of head injury, and not very often in depressed skull fractures. Trauma of these nerves occurs almost exclusively in penetrating injuries of the head

Trigeminal Nerve (Cranial V)

Both the gasserian ganglion and the primary divisions of this nerve — the ophthalmic, maxillary, and mandibular nerves — may be involved in head injury The ganglion may be injured by longitudinal fractures of the petrous bone extending into Meckel's cavity Involvement of the ganglion may result in serious and permanent functional loss, and in some instances paresthesias, at first there is severe facial numbness, to be replaced, as the numbness recedes, by crawling and pins-and needles sensations. The numbness and paresthesias are more severe than those which occur in patients after section of the sensory root for trigeminal neuralgia[24] Involvement of the maxillary nerve, which courses through the roof of the maxillary sinus to the infraorbital foramen, and of the infraorbital nerve which branches from it, is more common than that of other branches of the trigeminal. There is sensory loss in the side of the nose, the upper lip, and the cheek below the lower lid. The anterior 2 teeth or all the teeth on one side of the upper jaw may be numb depending on whether there is only infraorbital or a complete maxillary nerve involvement. The sensory loss from injury of the supraorbital nerve, a branch of the ophthalmic, is indicated by

numbness above the brow extending back to the coronal suture in a rectangular area from the midline to a line along the lateral border of the brow and the coronal suture line posteriorly. Fractures through the supraorbital and infraorbital foramens, or, more commonly, facial lacerations and periorbital trauma, are the usual causes of injury of the supra- and infraorbital nerves Restitution of function usually occurs within 6 months The mandibular nerve may be involved intracranially or with fractures of the lower jaw Depending on the site of involvement, there may be numbness of one-half of the lower jaw, the lower lip, and one-half of the anterior two-thirds of the tongue

Frontal fractures with involvement of the anterior ethmoid foramen may in rare instances injure the nasociliary nerve This may result in numbness of the inferior lateral aspect of the nose, and occasionally in conjunctival anesthesia

Facial and Acoustic Nerves (Cranial VII and VIII)

Complete and permanent facial paralysis after head injury is rare, and only in about 10 per cent of the patients with facial paralysis is there a permanent residual (Fig 48, *see also* Fig 101*A*). In complete unilateral facial paralysis, much more common than bilateral paralysis, there is· (*1*) inability to contract the facial muscles of expression, (*2*) inability to close the eye (paralysis of the orbicularis oculi); (*3*) widened palpebral fissure at rest; (*4*) smoothed out facial expression with drooping angle of the mouth (paralysis of orbicularis oris), (*5*) inability to wrinkle the forehead, (*6*) loss of the corneal reflex but not of pain sensation

Facial nerve trauma can occur with closed head injuries, with lacerations of the face and mastoid areas, with penetrating and perforating injuries of these areas, and with transverse fractures of the petrous bone These head injuries may be associated with bleeding from the ear(s), and a facial paralysis in such cases, appearing 4 to 12 days after the injury, may be due to edema or hemorrhage in the facial canal.

The site of the facial nerve trauma is of clinical importance If the

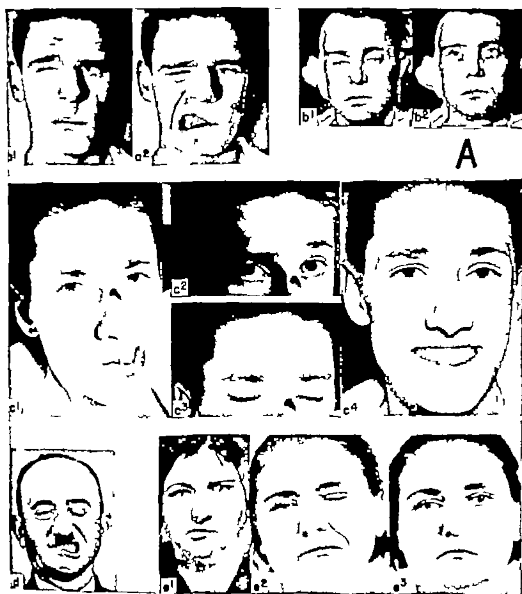


Fig 48. (*A*) Peripheral facial nerve paralysis in head injury (*a*) Involvement of forehead and of orbicularis oculi and oris muscles. (*b*) Bilateral paralysis patient trying to wrinkle forehead by looking upward (note lack of change) (*c*) Involvement of angle of mouth forehead and eye on right side recovery 6 weeks after injury (c^4) (*d*) Partial recovery with residual weakness and rigidity on right patient able to wrinkle forehead and close eye fairly well but angle of mouth does not rise properly (*e*) Left peripheral paralysis no improvement, 2 years after injury (e^1) and results of facial-spinal accessory nerves anastomosis (e^2–e^3) fairly good tone and some ability to contract facial musculature. (*Continued on next page*)



Fig 48 (*continued*) (*B*) Whiplash injury (hyperflexion), with "tear-drop" fracture of fifth cervical body (*a*) Soon after the accident (*b*) Healing 3 months later (*c*) Whiplash injury (hyperflexion), with spinal cord involvement and weakness of left upper limb and sensory dissociation of right side of body (Brown-Séquard syndrome).

lesion is in the portion of the nerve lying in the internal acoustic meatus, an associated involvement of the acoustic nerve is almost always present. The association of nerve deafness, a dead labyrinth, peripheral facial paralysis, dry cornea, and ageusia in the anterior two-thirds of the tongue on the ipsilateral side indicates clearly that the site of facial nerve injury is in the internal acoustic meatus Injury of the facial nerve peripheral to the chorda tympani, below the emergence of the chorda tympani nerve, does not affect the sense of taste or lacrimal function In most patients with facial paralysis after closed head injury, this is the site of nerve involvement; nevertheless, we have had 2 cases of involvement of the chorda tympani nerve in closed head injury without facial paralysis

Injury of the first portion of the facial nerve, with involvement of the stapedius muscle, will cause hyperacusis

In our entire experience we have seen only 3 cases of bilateral facial paralysis; 1 patient died, and in the other 2 the paralysis eventually improved. In one of our series, consisting of 425 patients with skull fracture, there were 14 cases of facial paralysis. Among the 667 patients

with skull fracture in our 1952 1954 series, facial paralysis occurred in 18.

Lacerations of the face are common in head injury In our 1952 1954 series, there were 439 patients with facial lacerations and contusions. We have seen 7 cases of facial paralysis associated with lacerations of the face, in 3 there was involvement of the pes anserinus, and in 4 of the facial nerve near the stylomastoid foramen.

Glossopharyngeal, Vagus, Accessory, and Hypoglossal Nerves (Cranial IX XII)

Trauma of these nerves occurs almost exclusively with penetrating head injuries seldom are they involved in the usual types of closed head injury, including skull fracture. We have seen 1 case in which cranial nerves III through XII were sheared off on one side by a missile which entered the skull at the base, and another case in which an occipital fracture due to a fall extended through the hypoglossal foramen, with unilateral paralysis of the hypoglossal nerve.

Uncal Herniation Syndrome (Figs 23, 25)

The earliest signs of uncal herniation *i.e* herniation of the temporal lobe and brain stem through the incisura, are occipital headaches and a clouding of consciousness in a patient with signs of increased intracranial pressure [22, 23] Other warning signs are (*1*) unilateral or bilateral pupillary inequality due to involvement of the third cranial nerve, (*2*) nuchal rigidity and paradoxic hemiparesis or hemiplegia, due to involvement of the peduncle (*3*) weakness or paralysis of upward gaze (*4*) visual disturbances, due to involvement of the posterior cerebral artery with infarction of the calcarine region. The clinical abnormalities in patients who survive uncal herniation are many and varied. Undue delay in the treatment of an uncal herniation may result in irreversible damage the patient is in deep coma the muscles are

flaccid, the swallowing reflex is lost, and there is an absence of nuchal rigidity.[18]

The clinical picture of herniation of the medulla and the cerebellar tonsils, an uncommon complication of acute head injury, is sudden arrest of respiration and continuing heart action[40]

Metabolic, Electrolyte, and Blood Changes

Some of the changes are the direct result of injury of the hypothalamic and pituitary areas, others the secondary manifestations of severe depletion states. Many of the metabolic disturbances which occur with head injury are transient and disappear without any treatment. In a series of 76 patients unconscious for 12 or more hours, with an overall mortality of 30 per cent, such disturbances were found in 90 per cent[28] *Proteinuria* occurred in the first 24 hours and gradually disappeared within 2 to 6 days The blood *urea* level was high in some, reaching a peak within 3 days and then subsiding. About 30 per cent had *glycosuria* of the low renal threshold type, and in some there was also *hyperglycemia* The *hyperchloremia* and *hyperchloruria* found in some patients Higgins and co-workers[28] attributed to injury of the inferior surfaces of the frontal lobes *Hypochloremia* and *hyperchloruria* developed in some cases 7 to 12 days after injury, particularly in the elderly patients; while the condition resembled Addison's disease, neither salt nor DCA or cortisone had any effect. In patients with brain stem trauma, hyperpyrexia and hyperpnea may have been the cause of respiratory *alkalosis*. The mortality in those with normal electrolyte balances was 24 per cent, whereas in the cases in which the abnormalities were of major character the mortality was 61 per cent; age, too was a factor in the mortality rate.

In our experience, most of the changes described above are caused by alteration in diet The symptoms and signs of electrolyte imbalance may be obscured by the unconscious state of a patient with severe head injury. In addition to the effects of the head injury itself, concomitant

factors, such as hyperpnea, increased intracranial pressure intracranial hemorrhage, cerebrospinal fluid leakage infections (meningitis, abscess) and injuries of other portions of the body affect the blood chemistry equilibrium

The concentrations of *sodium* and *potassium* in the blood may be abnormally high (hyperosmolarity) if the area of the preoptic nuclei and the pituitary gland or its blood supply are injured [12a, 34a, 60] In such cases, increased water intake does not affect the high concentrations.

Severe blows to the front or back of the head which involve the floor of the anterior fossa and the optic nerves and chiasm with possible injury of the blood supply of the pituitary gland and stalk, may cause *diabetes insipidus* It usually becomes manifest within 10 to 14 days and is occasionally a permanent complication [8, 24, 30a] In most cases however, there is improvement in the course of several months The condition is associated with polyuria and polydipsia the specific gravity of the urine is extremely low as low as 1.005 or less

Simmonds disease (hypopituitarism) after head injury although sporadically reported in the literature, has not occurred in any case in our experience.

Trauma of the central nervous system may in rare cases cause *gastro-intestinal hemorrhage* and *ulceration* or activate a pre-existing ulceration.[11, 17a, 20b]

Claude Bernard [2, 3] demonstrated experimentally that injury about the aqueduct of Sylvius and the fourth ventricle could cause glycosuria. Conceivably increased adrenal activity due to the stress of the head injury might also cause it. Hyperglycemia will also be evident if the stress of the head injury has caused a traumatic *diabetes mellitus*

Ketonuria is found occasionally this may be due to a change in carbohydrate metabolism as a result of the head injury or merely be the result of an inadequate diet.

Hypocalcemia and *calciuria* are common particularly in patients with associated fractures of long bones and therefore committed to prolonged bed rest

Ascorbic acid depletion occurs after trauma. It has been shown experimentally that some time is required after ascorbic acid is administered before the normal balance is re-established.[1, 7]

The proteinuria in head injury is largely the result of increased nitrogen excretion. This may be due to increased protein catabolism or to decreased protein utilization. The high fever which accompanies complications of severe head injury, *e.g.*, meningitis or abscess, plays a role in the increased catabolism.

Nutritional deficiencies may develop in the patient with head injury as a result of unconsciousness, vomiting, and diarrhea, the last-mentioned principally due to the faulty tube feeding.

Eosinopenia has been reported to be present in patients with head injury.[73, 75] There seemed to be some correlation between the severity of the injury, the clinical course, and the eosinophil count; 85 per cent of the patients had a lower eosinophil level in the circulating blood at first, with a return to normal as improvement set in. In 2 patients who died, the eosinopenia was sustained until death.

Whiplash (Hyperextension, Hyperflexion) Injury Syndrome (Fig. 48B)

Unconsciousness is rare in this type of injury, but the patient is frequently dazed and confused. Vertigo and nausea, the result of the subconcussive effects on the brain and labyrinths, are the common symptoms. In most patients, the symptoms are out of proportion to the findings, and may last for several weeks to several months. Women seek medical care for this condition more commonly than men. As in other types of injury, the nervous and anxious person has the most prolonged disability.

The most common complaints are of aching of the neck, with or without occipital headache. Onset of the neck pain and limitation of movement are frequently delayed for 24 to 48 hours, and a patient examined immediately after an accident may be discharged because

the roentgenograms do not show any injury Several days or weeks after the accident, however physical examination sometimes reveals limitation of movement in the cervical spine and particularly a difficulty in extending or flexing the neck. The posterior and lateral neck muscles are tender to palpation and may be in spasm. The results of neurologic examination are usually normal. The exact degree of injury which is confined to the muscles and ligaments is often difficult to establish With more serious injury, the pain in the neck radiates to the shoulder, arm and hand and there may be localized paresthesias in the thumb and index finger The neck pain may be constant and severe and may be increased by coughing Flexing the arm on top of the head will sometimes relieve the pain Since posturing of the neck may give some relief, a head tilt may result.

The symptomatology has been classified by some clinicians on the basis of the level of injury as "occipital" syndrome (radiation of pain into the occiput), with injury of the second posterior root lateral neck pain, with injury at the third and fourth vertebrae shoulder pain with injury at the fifth vertebra pain in the radial side of the hand with injury at the sixth vertebra pain in the index and middle fingers with injury at the seventh vertebra. Pain is sometimes felt over the precordium, simulating cardiac pain Injury of the long thoracic nerve may cause winging of the scapula

If there is associated involvement of the sympathetic trunk there may be pupillary inequality, and in some cases Horner's syndrome. Hearing deficits are uncommon Some blurring of vision, an early complaint disappears rapidly

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Chapter VI

SKULL FRACTURES

Skull fracture may be classified as linear or depressed, and simple or compound The linear fracture may be a single fracture line or a multilinear (comminuted) one. The simple fracture may be linear or depressed, but the overlying scalp is not lacerated, whereas in the open or compound fracture there is laceration of the scalp, or a communication created with such structures as the paranasal sinuses or the middle ear

Linear Fractures

Single linear fractures occur in a little over 80 per cent of the cases of skull fracture. These fractures are usually the result of elastic deformation of the calvarium (*see* Chapter III) About half of the fractures occur in the middle third of the skull and extend toward the middle fossa. The remaining half are about equally divided between frontal and occipital fractures.

In children all evidence of fine linear fractures usually disappears by the end of about 6 months, and in the very young both single linear and multilinear fractures usually heal very rapidly (*see* Fig 77) In

adults, a linear fracture may be visualized on the roentgenogram for several months to many years after its occurrence Healing of multilinear fractures which are accompanied by some comminution and some separation between the edges of fracture lines takes very much longer, in some cases as long as 8 to 10 years or more

Occasionally, there is loss of calcium along the edges of a linear fracture, so that several months after injury an area of bone loss may be found in the vicinity of the initial fracture line, both in the young and in adults Erosion of bone at the fracture site may be due to the presence of a leptomeningeal cyst Such a cyst may form if the fracture is associated with a dural and arachnoidal tear A tear of this type permits the formation of a cystic accumulation in the leptomeninges, cerebral pulsations then force additional cerebrospinal fluid into the cyst between the edges of the fracture, resulting in increasing erosion of the fracture border [10, 33, 35] Bone loss may also occur over a subdural hematoma[7, 28] or hygroma [30]

The prognostic importance of a fracture line is that it is a measure of the blow In an open linear fracture there is the possibility of infection entering the cranial cavity Linear fractures also may extend into the paranasal sinuses and the temporal bone, producing communications between the cranial cavity and the middle ear and sinuses despite the absence of lacerations of the overlying skin

Most linear fractures extend toward the base of the skull and thus help form basal fractures, but occasionally a small linear basal fracture may be unassociated with a fracture about the area of impact In such cases, there may be a sudden onset of meningitis during convalescence, due to the introduction of infection from the middle ear or the paranasal sinuses into the cranial cavity

Depressed Fractures

The clinical features of any depressed fracture depend upon. (*1*) site of the fracture; (2) type of depressed fracture, *i e.*, simple or com-

pound, penetrating or perforating, low velocity or high velocity injury, and so on, (3) amount of intracranial trauma Except in war injuries, there is often little clinical evidence of brain injury[4, 14]

There may be a scalp laceration overlying the depression (*see* Fig 86*B*), or, in some instances, at a distance from the depression (*see* Fig 87*A*) Severe hemorrhage may occur in some lacerations of the scalp Often, the scalp becomes discolored 2 or 3 days after the injury suggesting hemorrhage into its component parts. Hemorrhage into the subaponeurotic space may extend to the superciliary area anteriorly the superior nuchal line posteriorly, and the attachments of the temporalis muscle laterally In such cases, there is extensive swelling of the scalp

In about half the cases of depressed fractures, the dura is not torn. Penetrating and perforating fractures are accompanied by dural and cerebral trauma. The mechanisms of such injury are discussed in detail in Chapter III

About 50 per cent of depressed fractures are in the frontal area, the remainder in the parietal and posterior regions If the depressed fracture occurs over a venous sinus, the edges of bone may tear the sinus but the injury may be revealed only during the operative repair of the fracture when removal of the bone fragment is followed by hemorrhage from the sinus. Tears of the middle meningeal blood vessels and other dural vessels may also occur, with resultant hemorrhage (*see* Figs. 53-54)

The mortality rate appears to be related to the state of consciousness after injury Most patients who are conscious on admission to hospital and have no history of unconsciousness recover, whereas the mortality rate of those who remain unconscious is 35 per cent. The mortality among patients with dural tears and trauma of intracranial contents is much higher than among those without such involvement. Patients with tears of large venous and arterial channels present serious problems of surgical management and of morbidity and mortality

The neurologic examination of patients with depressed fractures

usually reveals little evidence of neurologic deficit except for an occasional aphasia and a hemiparesis or hemiplegia. When such localizing signs are present the morbidity and mortality are much higher than when they are absent.

In our large series, there were 637 cases of skull fracture, 40 of these were closed depressed fractures, 34 were open depressed fractures, 10 were open comminuted fractures, and 22 were closed comminuted fractures. There were 531 cases of single linear fracture. A summary of the data on 129 cases of depressed fractures is given in Table 6.

TABLE 6. *Findings in a Series of 129 Cases of Depressed Skull Fractures*

Finding	*Number of cases*	*Number fatal**	*Mortality rate, %*
Simple fracture	30	1	3.3
Open fracture	99	5	5
Dural tear	60	6	10
Unconsciousness			
Before hospital admission	56	1	1.7
Continued	14	5	35
None	59	0	0

* Total number of deaths, 6; mortality rate, 4.6 per cent.

Frontal Area

Open, depressed fractures in the region of the frontal and paranasal sinuses usually result from a blow to the forehead, as in automobile accidents, or with a penetrating or perforating fracture by bullet or shell fragment. Unconsciousness, pareses or paralyses, convulsive seizures, and focal phenomena are not usual features of frontal sinus fractures. Nevertheless, considerable involvement of the dura and brain may be found on operation. The cribriform plate, which is thin and vulnerable, is often shattered, and one or both hemispheral poles may be injured. The cribriform plate may also be injured by stresses arising in other parts of the head, not associated with the fracture at

the site of impact, these are the so-called "separated fractures, or indirect fractures.

Complications (pneumocephalus, meningitis, and brain abscess) are more apt to occur with fractures of the frontal area, but the immediate mortality rate is much lower than in fractures of the more posterior portion of the head

A fracture involving the frontal sinus may lead to the formation of a craniонasal fistula. Cerebrospinal fluid rhinorrhea is common in such cases.[1,2,6,8,10,26] In some individuals, the frontal sinuses communicate with each other, a fact to be remembered when rhinorrhea is present. The fistula may also result from a linear fracture elsewhere in the head (*see* p 212) A more or less frequent sequel of frontal sinus fractures is anosmia[23] particularly in patients with craniоnasal fistula and rhinorrhea. In some cases, the anosmia appears only after the fistula is repaired, due to injury of the olfactory filaments during the repair Patent craniоnasal fistula in the absence of obvious cerebrospinal fluid rhinorrhea, may be the cause of repeated attacks of posttraumatic meningitis.[20]

The paranasal sinuses may also be injured in basal fractures. In such cases, complicating infection such as meningitis or brain abscess may occur in the course of an apparently satisfactory convalescence.[23]

The presence of cerebrospinal fluid rhinorrhea may not be recognized while the patient is in bed If there is no recurrence after the patient has left the hospital, the fistulous tract probably has healed But if the rhinorrhea recurs, it is unlikely that the fistula will heal spontaneously For a discussion of cerebrospinal fluid rhinorrhea as a posttraumatic complication, *see* page 195.

Open fractures of the frontal sinus area may allow air to collect in the cranial cavity, under the skin of the forehead where it is disclosed by crepitation on palpation and even beneath the conjunctiva (*see* Fig 43)

Our findings in 54 cases of open fracture of the frontal area are summarized in Table 7

TABLE 7 *Data on Series of 54 Cases of Open Fracture of Frontal Sinus, Orbit, and Cribriform Plate Region*

Data	*Number of cases*
Deaths	4
Unconsciousness	
Before hospital admission	24
Continued	14
None	16
Dural tear	39
Complications*	
Cerebrospinal fluid rhinorrhea	11
Pneumocephalus	7
Brain abscess	3
Local infection	7
Meningitis†	6
Treatment	
Initial repair	47
Initial repair elsewhere	
Cranionasal fistula	6
Cranionasal fistula, extradural, subdural, and brain abscess	1

* None fatal except 1 case of meningitis
† In 3 cases, 4 or more attacks of meningitis

Orbital and Ocular Involvement (see Figs. 43-44)

Frontal blows, whether they result in fractures or not, may injure the orbit and eyes The injury ranges from massive destruction of the orbital contents, including the eye, to defects of vision or ocular movements, or simply to a periorbital hemorrhage, the common "black eye" Open fractures involving the orbit and eye are more common in warfare, and are more serious as a rule than those in civilian life In war injuries, one or both eyeballs may be destroyed, in addition to the cerebral and skull injuries of varying degree In civilian injuries,

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Carotid Artery-Cavernous Sinus Fistula (Fig. 49)

A blow to the frontal region, not necessarily severe, can disrupt the internal carotid artery in its course through the cavernous sinus and create a fistula. This complication of head injury is fairly rare, judging by our experience at the Detroit Receiving Hospital and at Grace Hospital, where we have seen 1 case each year for the past several years.

The early symptoms are orbital discomfort and headaches. Exophthalmos soon becomes evident and increases in severity, accompanied by slight to severe impairment of vision, and even blindness. The degree of visual loss usually depends on the rapidity of onset and the severity of the exophthalmos Conjunctival chemosis is found in severe cases. Papilledema and enlarged retinal veins are usually present, and auscultation of the area reveals a bruit and, later, pulsation of the globe. Both the bruit and the pulsation usually stop when the ipsilateral carotid artery is compressed, this serves as a diagnostic test. In persons in whom there is a communication between the cavernous sinuses, the other eye eventually also becomes involved

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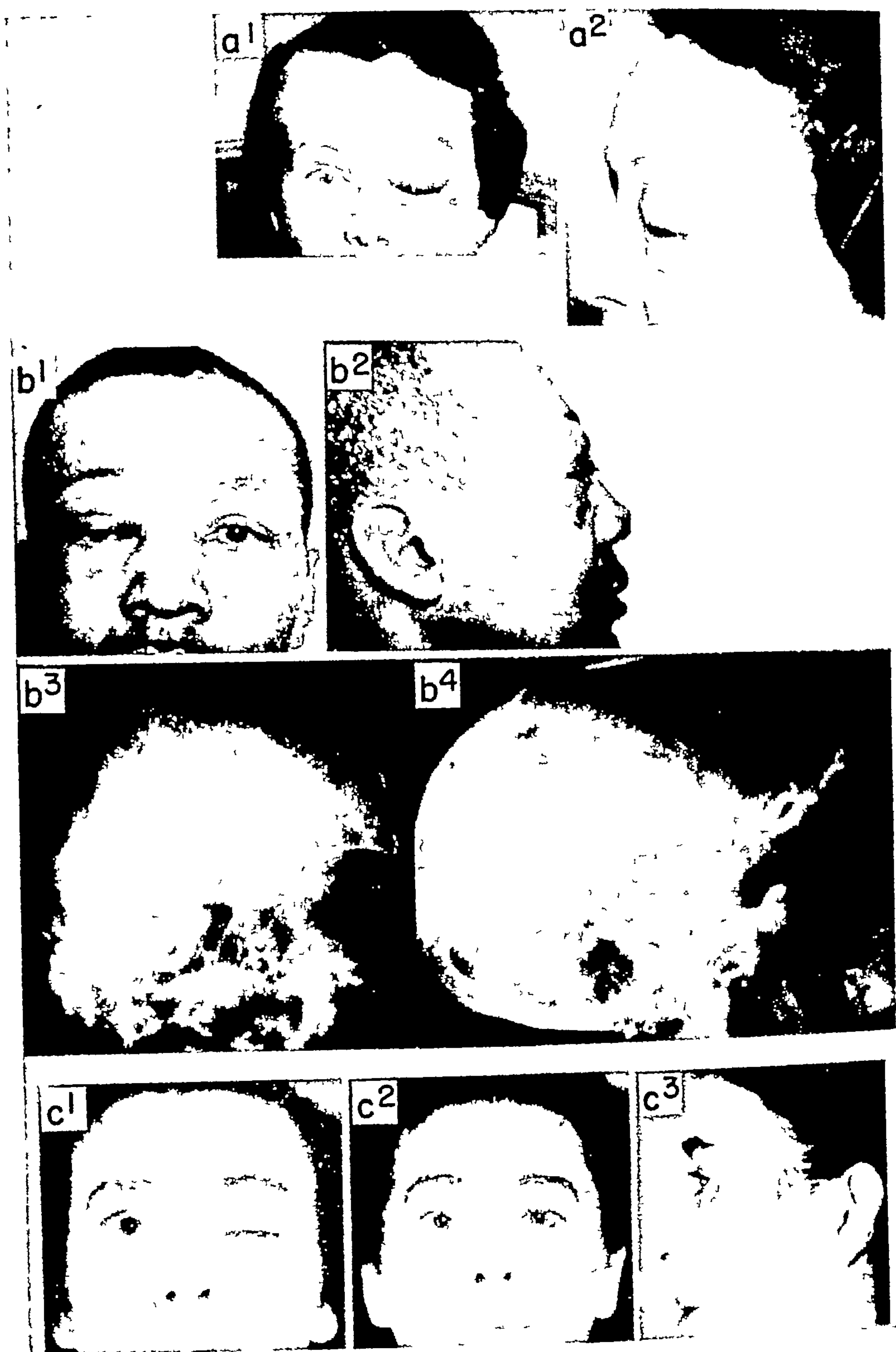


Fig 49 Carotid artery–cavernous sinus fistula (*a*) Left exophthalmos, bruit over left eye and left temple. (*b*) Right exophthalmos (*c*) Left exophthalmos; ligation of common carotid artery, with disappearance of exophthalmos.

It is not difficult to establish the diagnosis of the arteriovenous fistula provided the possibility of its occurrence is borne in mind in the presence of frontal injuries. Angiography is an aid. For best results, it is advisable to use the open method along with obliteration of the proximal portion of the carotid artery during the dye injection this assures a good dye concentration in the siphon and the orbital veins.

Temporal Bone and Hearing Apparatus Involvement

The temporal bone is fractured or injured by blows to the temporal and parietal, occipital and frontal regions. Since the bone houses the cochlear and vestibular organs and the facial nerve, the results of fracture may be serious complete or partial deafness, disturbed equilibrium and facial nerve paralyses Furthermore, if the dura is torn by the fractured bone, a pathway is created for the spread of infection from the ear to the intracranial structures The close proximity of the bone to the jugular bulb and the transverse, the sigmoid and the superior and inferior petrosal sinuses explains the extensive hemorrhage which may be associated with temporal bone fractures

In longitudinal fractures of the temporal bone, the following features are found, in various combinations (*1*) a fracture line coursing through the roof of the middle ear and along the anterior edge of the pyramid, through the superior wall of the eustachian tube and the carotid canal, (*2*) injury of the tympanic membrane, injury or fracture of the external canal walls, fracture of the ossicles, dislocation of the incus, and torn ossicular ligaments, (*3*) injury of the membranous cochlea by hemorrhage into the spiral ligament and the scala tympani (*4*) damage to the facial canal usually in the region of the genu (*5*) injury of the nerves to the internal ear by hemorrhage tearing or stretching (*6*) bleeding from the ear

In transverse fractures of the temporal bone, the features are (*1*) a fracture line coursing at right angles to the pyramid, (*2*) greater trauma of the cochlea than of the vestibule, (*3*) torn nerves in the internal auditory meatus (*4*) torn membranous labyrinth (*5*) hemor

rhage into endolymph and perilymph, (6) trauma of the middle ear, confined to the mesial wall; (7) facial nerve trauma, especially of the geniculate ganglion (over 50 per cent of cases), (8) hematotympanum, occasionally, (9) cerebrospinal fluid ottorrhea

Isolated fracture of the labyrinth is usually followed by complete loss of cochleovestibular function, but occasionally only the cochlea or the vestibule is fractured, with loss of function in the fractured part of the structure and partial or complete preservation of function in the other part

At autopsy soon after injury or many years later, the same changes have been found stretched and torn nerves, and signs of hemorrhage, particularly in the perilymphatic spaces of the basal coil of the cochlea [18]

Severe bleeding into the labyrinth is likely to be followed by considerable connective tissue organization of the blood clots, which is later replaced by formation of new bone within the osseous labyrinth

Fractures of the labyrinth heal with new bone formation in the endosteal and periosteal layers, and with connective tissue in the endochondral layer Healing is fairly well advanced within a month of injury in most cases, but occasionally the fracture heals incompletely or not at all and can be seen on roentgenograms many years later. In such cases, it is conceivable that a middle ear infection could reach the meninges

Deafness and vestibular disturbances may also occur in persons with head injuries in whom there is no clinical or roentgenographic evidence of skull fracture (*see* p 87).

The incidence of ear involvement varies in different series: (*1*) of 152 cases of head injury examined within 24 hours of injury, 49 had symptoms or signs referable to the [illegible] 50 per cent of another series had traumatic changes,[28a] (3) [illegible] of acute head [illegible] 129 had bleeding from the ears,[18a] [illegible] cases, 33 per [illegible] demonstrable hearing loss.

Penetrating and Perforating Fractures[11, 13, 18, 21, 24, [illegible], [illegible], 40]

The common head injury in war is the penetrating or perforating fracture, with the associated craniocerebral trauma (Fig 50, *see also* Figs. 92, 93) Unlike the penetrating fractures of civilian life (Fig 51), which are for the most part caused by a large variety of low velocity missiles and implements, the war injuries are largely caused by high velocity missiles—bullets and shell fragments. Because of the physical forces involved, the perforating fracture frequently causes death immediately or soon after injury On autopsy, extensive necrosis and destruction of cerebral tissue in the bullet's tract is found

The signs and symptoms of a penetrating fracture vary with the extent of craniocerebral involvement, which in turn depends on the injuring object and its velocity Penetration by a sharp object such as a knife or stiletto despite the cerebral trauma it causes, as indicated by neurologic manifestations does not necessarily cause unconsciousness. A high velocity bullet or shell fragment, on the other hand may cause severe cerebral trauma and many who sustain such an injury may be unconscious.

Patients with penetrating wounds can be divided into three groups (*1*) those who are in deep coma from the beginning and show signs of shock (2) those who are initially semiconscious but whose state deteriorates, and (3) those who remain conscious. In the first group, the blood pressure may be near normal levels, but the skin is of a grayish yellow pallor and respiration is labored or of the Cheyne Stokes type Air hunger is manifest in some, and death occurs within a few minutes to an hour or two in spite of all resuscitative measures. The second group although initially semiconscious and apparently not in a serious state of shock with normal respiratory rate, and responding to painful stimuli and even to speech, may nevertheless soon slip into coma breathing becomes more rapid until there is hyperpnea, secretions collect in the upper respiratory tract, the temperature rises

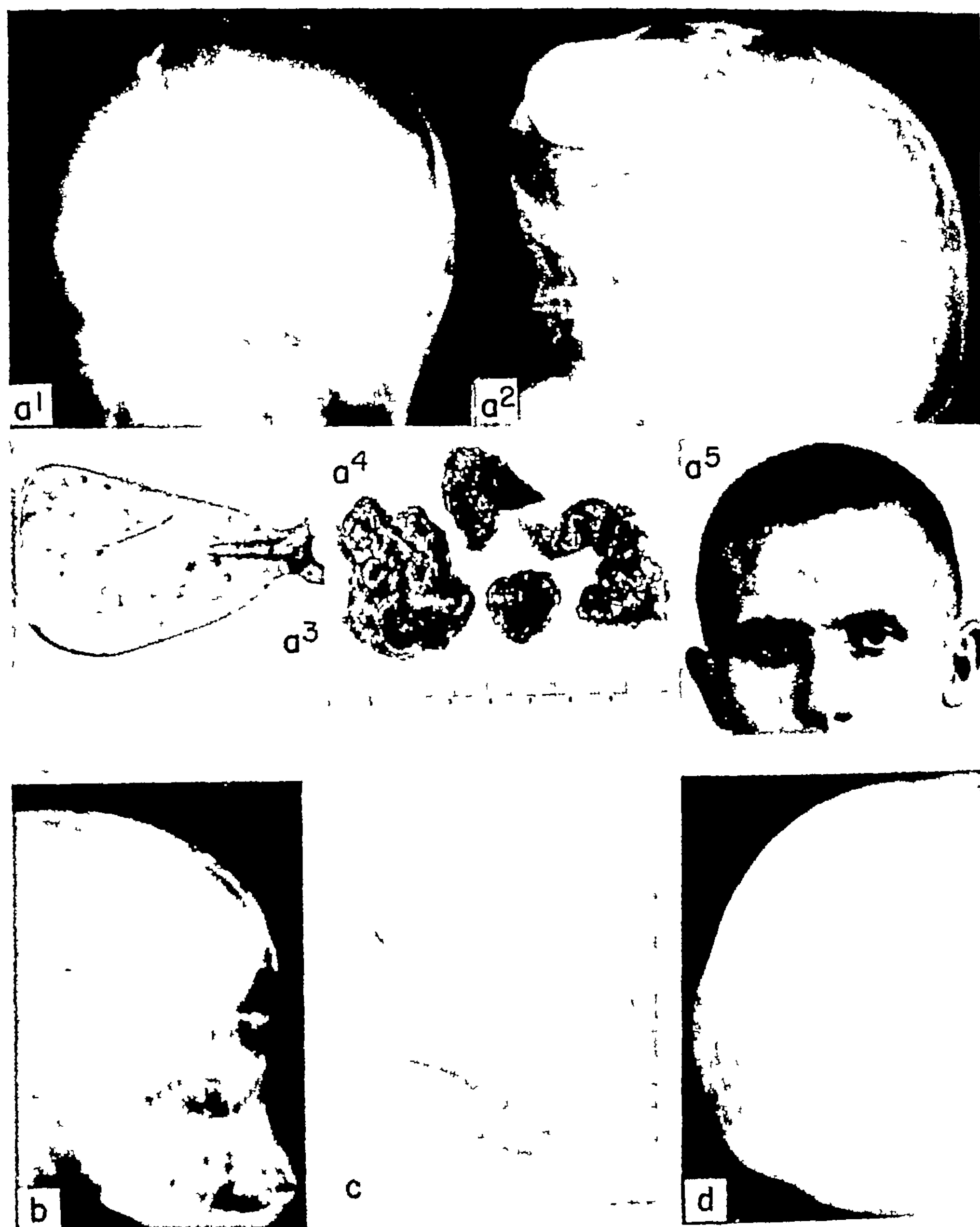


Fig 50 Penetrating injuries by various objects (*a*) By broken-off automobile fan blade preoperative roentgenograms (a^1–a^2), blade removed at operation (a^3), 30 Gm hematoma removed from right frontal lobe (a^4), and postoperative appearance of patient (a^5), during operation patient became increasingly drowsy, but awoke as hematoma was removed, posttraumatic epilepsy fairly well controlled with anticonvulsants (*b–d*) Foreign bodies in skulls and dura, usually removed by extracting a button of bone around the object by means of trephine, then removing the object from the bone and replacing the bone in the skull, (*b*) knife blade embedded in skull for 25 years.

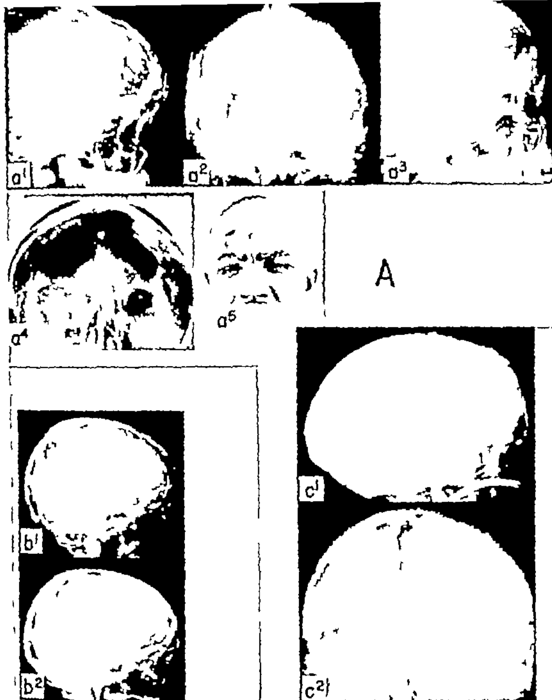


Fig 51 Penetrating head injuries by bullets in civilians all healed by primary intention. (*A*) Injury in right frontotemporal region (*a*) primary debridement delayed for 72 hours dura badly torn, brain necrotic, most of frontal lobe pulped (a^1–a^3) postoperative appearance (a^4–a^5) frontal lobe excised completely by suction, dural defect repaired with temporal fascia so as to separate nasal cavity from dural sac, wound closed without drainage skull defect repaired later with tantalum (*b*) Tangential injury of right parietal area and its repair extensive bone fragmentation dural laceration, and cerebral laceration 10 hours after injury extensive debridement, excision of pulped cerebral tissue, and repair of dural defect with fascia lata skull defect later repaired with tantalum (*c*) Explosive head injury by traversing bullet only a few small fragments of lead found the main mass of bullet having escaped debridement of areas of entrance and exit, both rather small removal of all pulped tissue in bullet tract repair of dura operation 14 hours after injury (*Continued on next page*)

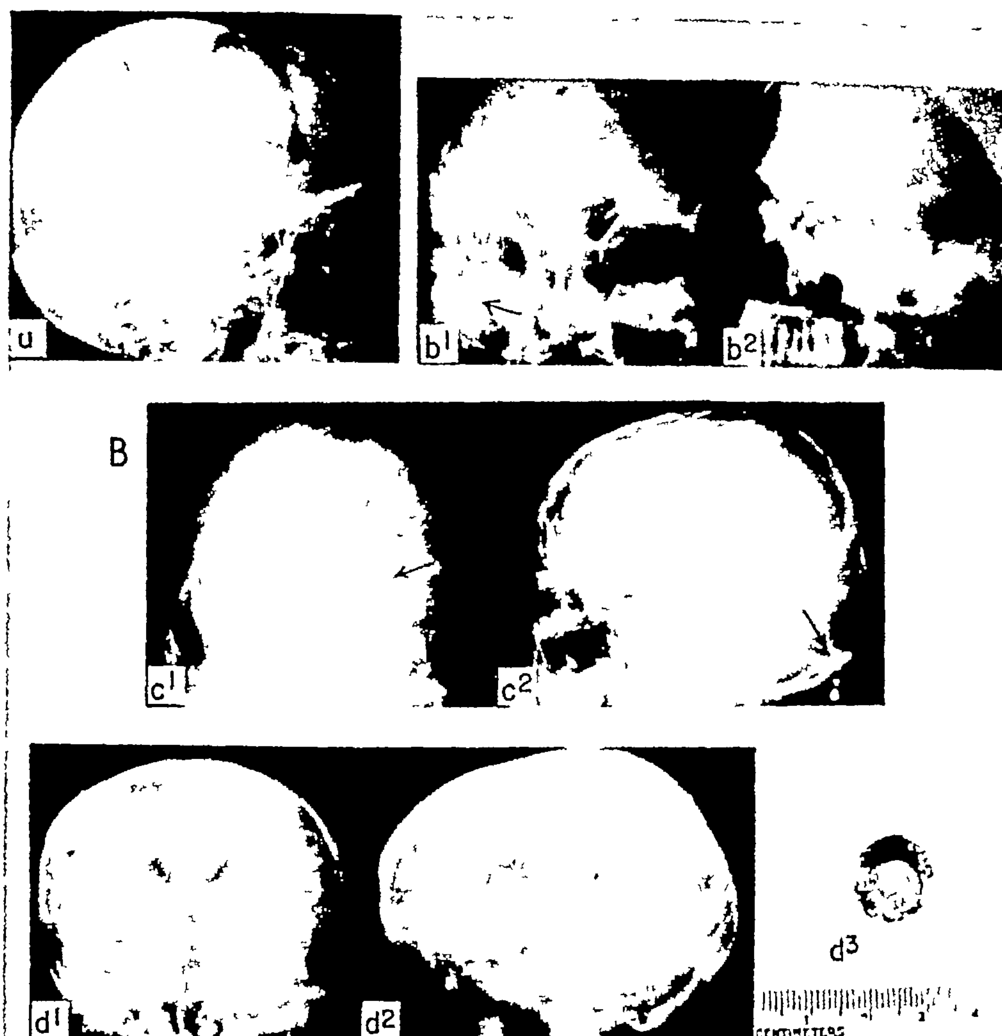


Fig 51 (*continued*) (*B*) Explosive injury of left frontoparietal area (*a*), wound of exit in forehead, debridement 26 hours after injury, healing by primary intention (*b*) Bullet entered head through ear, cerebrospinal fluid otorrhea and leakage of cerebral tissue from right ear, bone fragments, bullet fragments, and necrotic temporal lobe removed dura repaired (*c*) Tear of right lateral sinus by bullet fragments, repaired by piece of muscle inserted into tear (*d*) Visual disturbances and papilledema 2½ years after bullet injury, no cyst around bullet, decompression over right posteroparietal area caused increasing herniation at site; bullet removed through transcortical incision, dura repaired with fascia lata; pressure phenomena disappeared as operative area healed.

steeply, and death occurs within 12 to 18 hours The third group, also apparently severely injured, does not become unconscious and the vital functions remain normal; the likelihood of recovery with routine management in such cases is usually excellent. The difference between

the first and third groups is the degree of involvement of brain stem centers, involvement in the former being severe

In some, there are major neurologic deficits convulsive seizures, contralateral hemiparesis or hemiplegia, partial or complete blindness, speech disturbances. In others, there is a remarkable absence of focal abnormalities.

Not all injuries by missiles include dural penetration. In some cases only the scalp is extensively lacerated,[25] in others there is extensive comminution of bone In such cases, the possibility of an extradural or a subdural hematoma or a subdural collection of cerebrospinal fluid must be borne in mind

In suicide attempts, the gun muzzle may be directed forward in the temporal area, resulting in fracture through the anterior bony structures of the face, just below the anterior fossa, with unilateral or bilateral involvement of the orbit and optic nerve. Despite extensive injury of bone and soft tissue, these patients are usually conscious on hospital admission. Extradural and subdural hemorrhages are possible.[6, 20]

Dural lacerations usually occur with bursting fractures, with the tangential[8] or gutter type of injury, and with transventricular or other perforations, in short, with any head injury in which bone fragments can be driven in. In the gutter type of injury there is usually extensive damage of scalp, skull and brain, and a good likelihood of infection and brain abscess.[22, 31, 29]

Metal missiles cause deep tract damage and carry organic material on their surfaces. Jagged missiles are more damaging than smooth ones. Bullets are much cleaner than shell fragments.

Survival after ventricular injury was reported in many cases of penetrating fractures in World War II[27] In a few cases, the bullet was free in the ventricular cavity, moving about with head motion, successful treatment was reported in some of these cases.[12, 14, 17]

Any of the cranial venous sinuses may be injured but particularly the superior sagittal and the transverse sinuses.[27] The orbital region is

injured frequently,[14] and injury of the cerebellum may occur.

Extradural, subdural, and intracerebral hematomas are common complications,[6, 26] and in some cases there are intraparenchymatous clots in the cerebellar lobes as well.[3] The hematoma is usually at the site of the penetrating fracture, but contralateral subdural and subcortical hematomas have also been found.[26] Hematomas usually occur in the distal portion of the missile tract, even when the foreign body has traversed the midline.[27] Hematomas may also occur with tangential injury of the scalp and skull, and they may be extradural, subdural, or subcortical.[9] Of 316 patients with penetrating head injuries treated within 8 hours of injury by an Army Neurosurgical Unit, 46 per cent had complicating intracranial hematomas; the percentage increased as the time between injury and treatment lengthened.[1]

In the *diagnosis* of a penetrating or perforating head injury, particular attention in the examination must be directed to: (*1*) The presence of contusions and lacerations of the head and injuries elsewhere in the body, since even the smallest laceration may be significant, small lacerations in the back of the neck and head may point to extensive basal and occipital involvement. (2) The state of the vital functions (blood pressure, pulse, temperature, and respiration), since these may provide a clue, particularly the temperature, to the possible presence of intracranial infection. (*3*) The neurologic status, which should be carefully evaluated and recorded, since this provides a basis of comparison, should changes occur later. All the diagnostic technics may have to be drawn on, and should be carefully executed.

The *prognosis* for patients with penetrating head injuries depends on the initial effects of the injury, hemorrhage in the missile tract or after initial debridement, and posttraumatic infection or other complications. Recovery of function depends on the area damaged and the extent of the damage. Left-sided lesions involving the motor cortex and the speech areas may be associated with possibly permanent severe speech defects and a right hemiparesis or hemiplegia.

Homonymous visual field defects may result from extensive injury of the occipital region and these too may be permanent. Mental and psychologic abnormalities, if present, are probably proportional to the destruction of cerebral tissue, the greater the amount of tissue destroyed, the greater the likelihood of considerable abnormality. Posttraumatic epilepsy is much more common with penetrating head injuries than with closed ones, occurring in about 50 per cent of cases followed for 10 years or more. In one large series of penetrating injuries, followed for 5 years, 43 per cent had posttraumatic epilepsy.[22]

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injured frequently,[34] and injury of the cerebellum may occur Extradural, subdural, and intracerebral hematomas are common complications,[6, 26] and in some cases there are intraparenchymatous clots in the cerebellar lobes as well[3] The hematoma is usually at the site of the penetrating fracture, but contralateral subdural and subcortical hematomas have also been found[26] Hematomas usually occur in the distal portion of the missile tract, even when the foreign body has traversed the midline[25] Hematomas may also occur with tangential injury of the scalp and skull, and they may be extradural, subdural, or subcortical[9] Of 316 patients with penetrating head injuries treated within 8 hours of injury by an Army Neurosurgical Unit, 46 per cent had complicating intracranial hematomas, the percentage increased as the time between injury and treatment lengthened[3]

In the *diagnosis* of a penetrating or perforating head injury, particular attention in the examination must be directed to (*1*) The presence of contusions and lacerations of the head and injuries elsewhere in the body, since even the smallest laceration may be significant, small lacerations in the back of the neck and head may point to extensive basal and occipital involvement (2) The state of the vital functions (blood pressure, pulse, temperature, and respiration), since these may provide a clue, particularly the temperature, to the possible presence of intracranial infection (*3*) The neurologic status, which should be carefully evaluated and recorded, since this provides a basis of comparison, should changes occur later All the diagnostic technics may have to be drawn on, and should be carefully executed

The *prognosis* for patients with penetrating head injuries depends on the initial effects of the injury, hemorrhage in the missile tract or after initial debridement, and posttraumatic infection or other complications Recovery of function depends on the area damaged and the extent of the damage Left-sided lesions involving the motor cortex and the speech areas may be associated with possibly permanent severe speech defects and a right hemiparesis or hemiplegia

Homonymous visual field defects may result from extensive injury of the occipital region, and these too may be permanent. Mental and psychologic abnormalities, if present, are probably proportional to the destruction of cerebral tissue the greater the amount of tissue destroyed, the greater the likelihood of considerable abnormality Post traumatic epilepsy is much more common with penetrating head injuries than with closed ones, occurring in about 50 per cent of cases followed for 10 years or more. In one large series of penetrating injuries, followed for 5 years, 43 per cent had posttraumatic epilepsy [32]

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Chapter VII

VASCULAR LESIONS, HEMORRHAGES, AND HEMATOMAS IN HEAD INJURY

Intracranial Vascular Lesions (Figs 52-63)

Traumatic intracranial vascular lesions include hemorrhage, thrombosis, and infarction. Hemorrhage may be classified as extradural, subdural (acute, subacute, chronic) subarachnoid and intraparenchymatous (massive or petechial) Thrombosis of arteries and veins may occur in the neck at the base of the skull, or intracranially In farction may result from thrombosis of a large vessel, contusions, or a tear in a blood vessel with occlusion and thrombosis.

Vascular lesions in craniocerebral trauma may be primary or secondary depending on whether they are caused at the time of the impact or result from the secondary effects of the injury Primary lesions include tears and contusions of any of the blood vessels in the skull dura, and brain. The intracranial mass lesions are the results of such primary lesions, with involvement of the sinuses, the middle meningeal artery and veins, and the cerebral connecting veins and vessels Contusions of vascular walls, eventually resulting in a thrombosis are also considered primary lesions.

A thrombus may occur in the carotid artery in the neck or at the

base of the skull, and in the middle cerebral artery; Verneuil[90] was the first to describe this Other branches of the circle of Willis, too, may be involved in basal injuries The internal carotid artery may be injured by fractures of the middle fossa in the base, it can also be

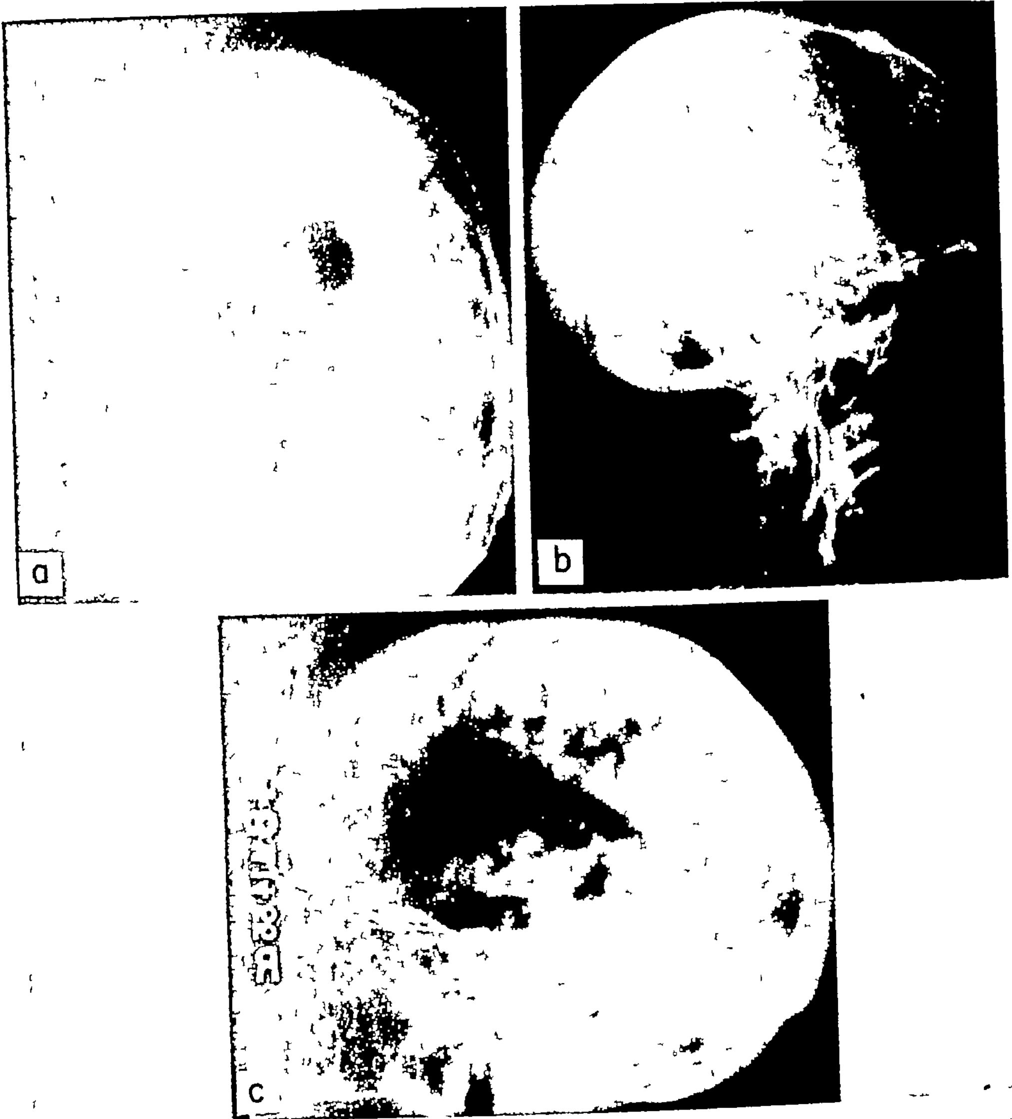


Fig. 52. Vascular lesions in head injury Patient became paralyzed immediately after hospital admission, but his condition improved despite the paralysis, tenta tive diagnosis of thrombosis of middle cerebral artery branches, with resultant cerebral infarction, was confirmed by finding an almost complete occlusion of branches of left middle cerebral artery, pneumoencephalograms 2 years later show relatively larger left ventricle, enlarged sulci and subarachnoid spaces, and exten sive cerebral atrophy.

involved in its intracranial course along with the middle cerebral artery Since head and neck injuries commonly occur together, thrombosis of the internal carotid artery is possible after direct blows to the neck or lower jaw, whiplash injuries of the neck or head, or accidents which twist or stretch the neck.[12, 22, 28, 42, 75, 85] Uncal herniation or localized cerebral swelling due to temporal lobe contusions can compress the artery, the swelling may also cause kinking of the artery at its junction with the middle cerebral artery The clinical picture of an extending and progressive thrombosis of the arteries at the base is paralysis and a seriously impaired state of consciousness, or a severe hemiplegia with an improving state of consciousness. Although thrombosis of the internal carotid artery is a rare complication of closed head injuries, its possibility should be kept in mind (Fig. 52)

Involvement of the anterior half of the sagittal sinus usually does

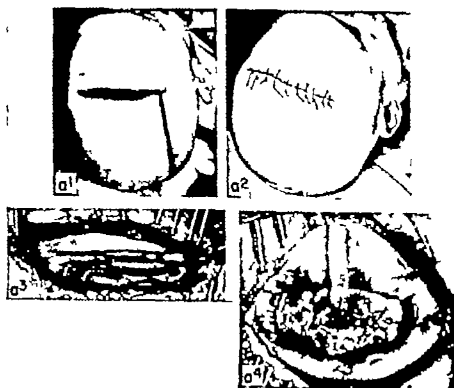


Fig 53 Vascular lesions in head injury Tear of sagittal sinus by open depressed midline fracture bleeding controlled by Oxycel, tear repaired by small muscle stamp.

not cause any symptoms or signs Involvement of the posterior half, with thrombosis, results in bilateral motor cortex dysfunction, leading to triplegia or quadriplegia Tears and occlusion of the sinus more posteriorly, near the confluence of sinuses, usually do not cause a motor deficit, but there is frequently a homonymous hemianopsia or complete blindness Lateral sinus injuries usually are not associated with serious signs, and the sinus may be ligated Increased intracranial pressure and papilledema may result from lateral or superior sagittal sinus involvement with occlusion A primary injury of a venous sinus, which itself might not cause any difficulty, may nevertheless result in thrombosis of the sinus with grave consequences (Figs 53-54)

Secondary vascular effects are usually due to increased intracranial pressure from cerebral edema or from intracranial mass lesions Compression or kinking of intracranial blood vessels may cause extensive infarction in their distribution Cerebral infarction occurs frequently in craniocerebral trauma This may involve large areas of the hemisphere on the affected side The junction of the middle cerebral and the internal carotid arteries may be compressed by mass lesions or by extensive contusion with edema The anterior cerebral artery and its branches may be compressed as they turn caudally to the straight gyrus near the border of the falx The superior cerebellar arteries may be compressed by the tentorial border, and the posteroinferior cerebellar artery may be involved at the foramen magnum if there is cerebellar herniation at this site

Uncal herniation, by compressing one or both posterior cerebral arteries,[32, 63] may cause infarction in the distribution of these vessels and, if the calcarine arteries are also injured, involve the visual pathway Many or all the branches of the posterior cerebral artery may be involved and compressed. Distortion of the brain stem, with downward movement of the brain stem and cerebellum, puts stress on perforating vessels to the brain stem and results in hemorrhage within it These perforating vessels are branches of the posterior cerebral and the basilar arteries Compression of the veins in this vicinity may also

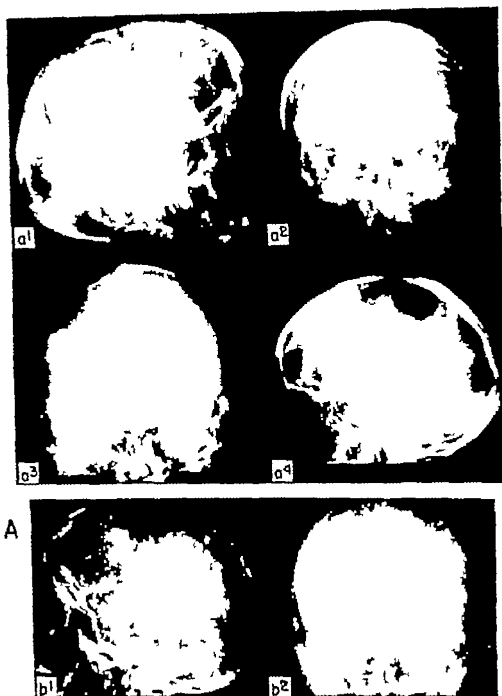


Fig. 54. Vascular lesions in head injury. (A) Laceration of posterior third of sagittal sinus by depressed fracture (a) lateral aspect of sinus sutured to re-establish patency, hemostasis obtained, and patient's recovery satisfactory; preoperative (a^1–a^2) and postoperative (a^3–a^4) roentgenograms. (b) Laceration of anterior portion of sagittal sinus by depressed fracture, repaired by double ligation, a harmless procedure in this part of sinus. (Continued on next page)

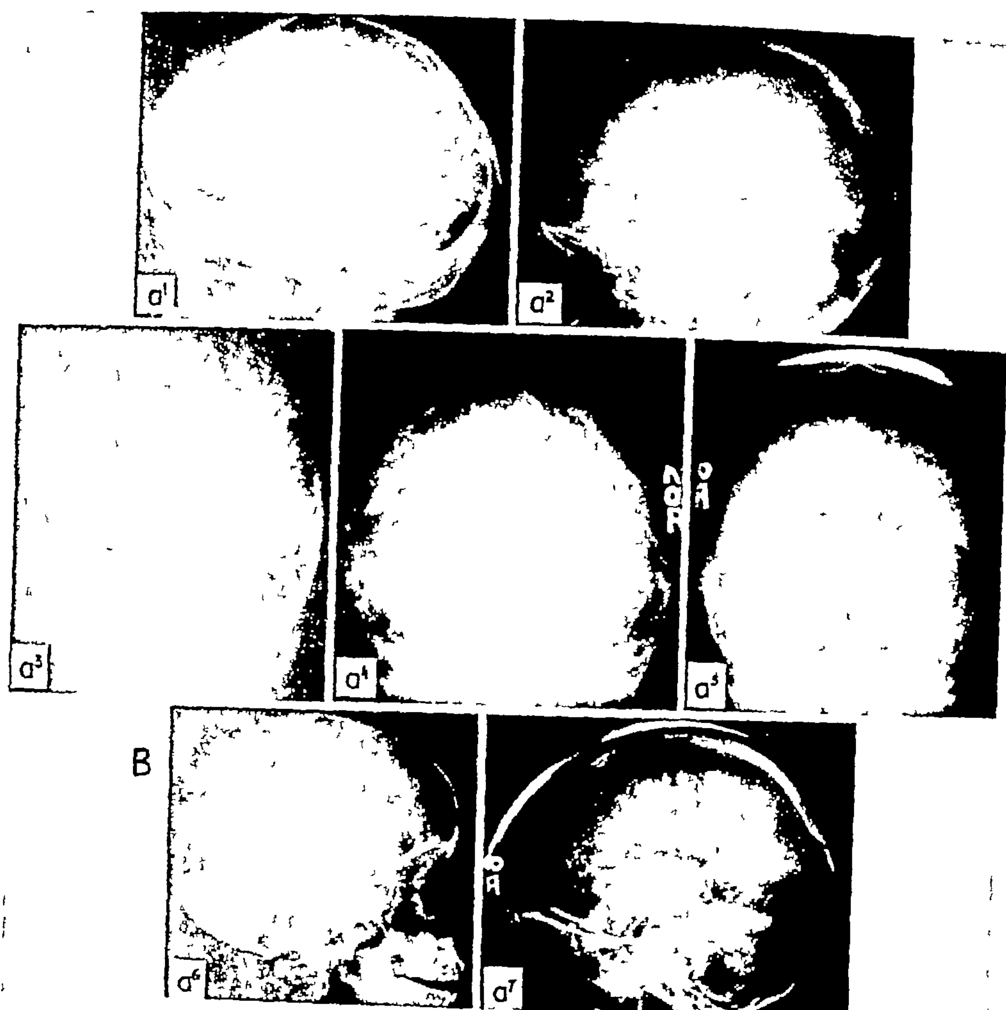


Fig. 54 (*continued*) (*B*) Tear of lateral sinus by depressed fracture of occipital bone (a^1, a^3), repaired by double ligation, postoperative roentgenograms (a^2, a^4), and pneumoencephalograms 6 months later (a^5, a^7), occipital horn of left ventricle somewhat enlarged, suggesting communicating porencephalus, undoubtedly due to contusions of left occipital hemisphere, tantalum cranioplasty for skull defect (a^6).

lead to hemorrhagic areas in the brain stem as a result of ischemia and hemorrhage by diapedesis Uncal herniation may also involve branches to the posterior hypothalamus from the posterior cerebral artery, so that the hypothalamus and mammillary bodies may be the sites of hemorrhage as the result of ischemia and infarction. Compression of the anterior choroidal artery by the anterior portion of an uncal herniation may cause a hemorrhagic infarction in the region

of the globus pallidus, as has been found on autopsy Distortion of the blood supply into the pituitary gland may cause infarctions in this structure, along with vessel wall injury and hemorrhages, traction on the branches supplying the infundibular region, due to lateral displacement of the midline structures by mass lesions, may be a factor Distortion of the optic chiasm, with hemorrhages and areas of infarction, has been described

Infarction due to thrombosis of the middle cerebral or the internal carotid artery may, if the patient survives, result in hydrocephalus ex vacuo. Cerebral ipsilateral edema may be associated with an extensive area of infarction, with a resultant shift of vessels on the angiogram or a shift of ventricles on the ventriculogram, suggesting a mass lesion.

Extensive infarction may result from deep cerebral contusions, particularly in the frontal and temporal lobes, and from hemorrhagic areas. Contusions cause the death of cells by disrupting their blood supply and nutrition, small hemorrhagic areas, as in petechial masses, may destroy neural tissue, with eventual formation of cystic cavities, the size depending on the size of the hemorrhagic area.

Subarachnoid and Pial Hemorrhages

Such hemorrhages are usually due to direct blows or intracranial mass movements, and are a common accompaniment of craniocerebral injury They are found in over 80 per cent of cases that come to autopsy, and may be present in as high as 50 per cent of the clinical material if bloody cerebrospinal fluid is taken as evidence. However, it should be remembered that blood in the cerebrospinal fluid may be present not only as a result of tears in the piarachnoid but also of contusions and lacerations of the cerebral surface or of penetrating cerebral wounds. In an occasional case of intracerebral hematoma, blood may escape through an opening in the cerebral surface to the subarachnoid and subdural spaces. At autopsy or on surgical exploration, small collections of clotted material may sometimes be found in the crevices of the subarachnoid space, or there may be a suffusion of blood in the

piarachnoid. The concentration of blood in the subarachnoid space may be much higher than that in the cerebrospinal fluid obtained by lumbar puncture

Subarachnoid hemorrhage may simulate a dynamic lesion, with progressing stupor and increasing hemiparesis or hemiplegia On the other hand, the signs and symptoms of subarachnoid hemorrhage may be simulated by hemorrhage elsewhere in the head Lumbar puncture may lead to improvement in some cases of subarachnoid hemorrhage, but in others surgical exploration becomes necessary In a series of 67 patients, among the indications for such exploration were (*1*) unilaterally dilated pupil, (2) unilaterally dilated pupil, with contralateral paralysis, (*3*) hemiparesis or hemiplegia, (*4*) a lucid interval, (*5*) continued unconsciousness, with disorientation, and (*6*) convulsive seizures Exploration revealed that in some patients contusions of the cerebral surface and cerebral edema were the causes of the clinical picture

Angiography should obviate the necessity for surgical exploration in most such cases However, cerebral edema may cause a shift of intracranial structures in the angiogram or pneumoencephalogram, and misdiagnosis is then possible

The irritating presence of blood in the subarachnoid space causes an inflammatory reaction and, eventually, piarachnoidal adhesions In some cases these may be so extensive as to produce obstructive hydrocephalus Usually, however, subarachnoid hemorrhage seems not to be followed by any sequelae

Extradural Hematoma[34, 68, 93, 98]

The extradural hematoma (Figs 55-57) is a collection of blood between the skull and the dura as a result of bleeding from injured extracerebral blood vessels These may be the middle meningeal artery and veins, the dural sinuses (the superior sagittal and transverse sinuses particularly), and the small connecting vessels between the dural surface and the diploic veins Extradural bleeding may also occur when

the dura is separated from the bone as a result of blunt head injuries without skull fracture,[7] as the blood collects it causes further separation and increasing exposure and tearing of blood vessels.

TABLE 8. *Data on 56 Cases of Extradural Hematoma of Middle Meningeal Origin**

Data	*Number of cases*
State of consciousness	
Lucid interval	25
Continued unconsciousness	18
Drowsiness and disorientation	11
Brief unconsciousness	2
Condition of pupils	
Dilated, ipsilateral	34
With contralateral hemiparesis or hemiplegia	32
Dilated, contralateral	4
Equal	18
Extraocular palsies	
Cranial nerve III	6
Cranial nerve IV	1
Cranial nerve VI	1
Divergent strabismus	1
Hemiparesis or hemiplegia	
Contralateral	44
Homolateral	3
Cerebrospinal fluid features	
Pressure up to 650 mm H_2O	28
Bloody	25
Clear	3
Associated mass lesions	
Subdural hematoma	9
Subdural hygroma	3
Clot in temporosphenoidal lobe	4

* Recovered 36 cases died 20 cases Sex 51 male, 5 female.

Extradural hematoma is a complication in 1 to 25 per cent of patients with head injury[25, 67] It usually grows large enough to cause signs of compression within several hours, but even a fairly large clot may cause no signs for a week or more, and possibly for as long as a month[45, 62, 81, 88] The small clots arising from diploic vessels are as a rule of no clinical significance, occasionally they are found during operative repair of a skull fracture However, large hematomas from this source have been reported[93] Subdural hematoma or subdural collection of cerebrospinal fluid (hygroma) may also be present, as well as intracerebral hematoma Findings in a series of 56 cases of extradural hematoma are summarized in Table 8

The mechanisms of injury of extracerebral vessels are discussed in detail in Chapter III Briefly, blunt low-velocity blows to the head, such as suffered in falls or in fighting, and penetrating injuries, usually of low velocity, are the most frequent causes of extradural hematoma[36] Linear fracture is frequently found in connection with extradural hematoma

Pathology

As found on operation, the hematoma is often a discus-like mass, weighing between 25 and 100 Gm (Fig 55*A*) Much larger masses have been found at autopsy, the weights ranging from 200 to over 300 Gm, and even as high as 450 Gm[88] The most usual location is in the parietotemporal area, sometimes extending over the occipital region and as far as the posterior fossa and the cerebellar lobe,[78] the frontotemporal, parieto-occipital, and frontal areas are other sites, in that order of frequency Extradural hematomas in the posterior fossa were believed to be uncommon,[54, 88] but many cases have been reported in the last 15 years[2, 5, 8, 14, 18, 20, 30, 35, 40, 49, 68, 71, 83]

Several days after the start of hematoma formation, a thin layer of the clot has become absorbed, so that the dura is thickened and there is a mass of clotted blood attached to the dural surface If the clot is

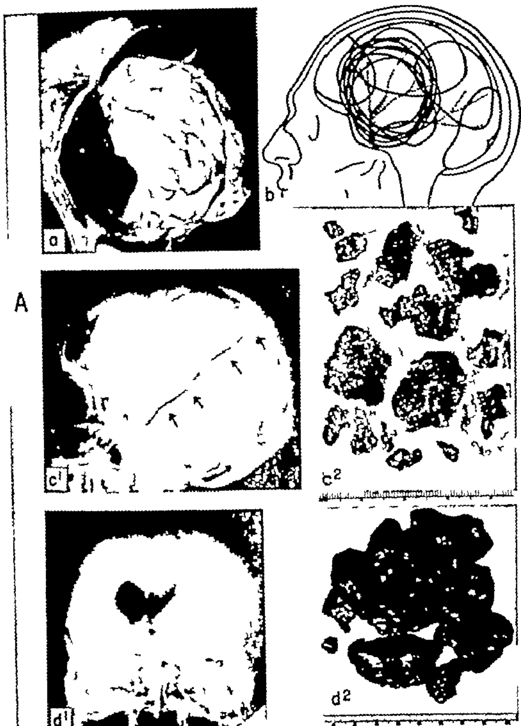


Fig 55 Extradural hematoma of middle meningeal origin. (*A*) Hematoma (*a*) found at autopsy of boy who fell and died in his sleep 5 hours later no associated cerebral damage found child could have been saved had the condition been recognized. (*b*) Composite diagram of 11 cases of extradural hematoma in 151 consecutive cases which came to autopsy most are in temporofrontoparietal area, a few in frontal some in posteroparietal and some close to sagittal sinus. (*c*) Hematoma with fracture of parietotemporal area. (*d*) Hematoma (pneumoencephalogram and 80 Gm. hematoma) in patient suspected of having a mass lesion. (*Continued on next page.*)

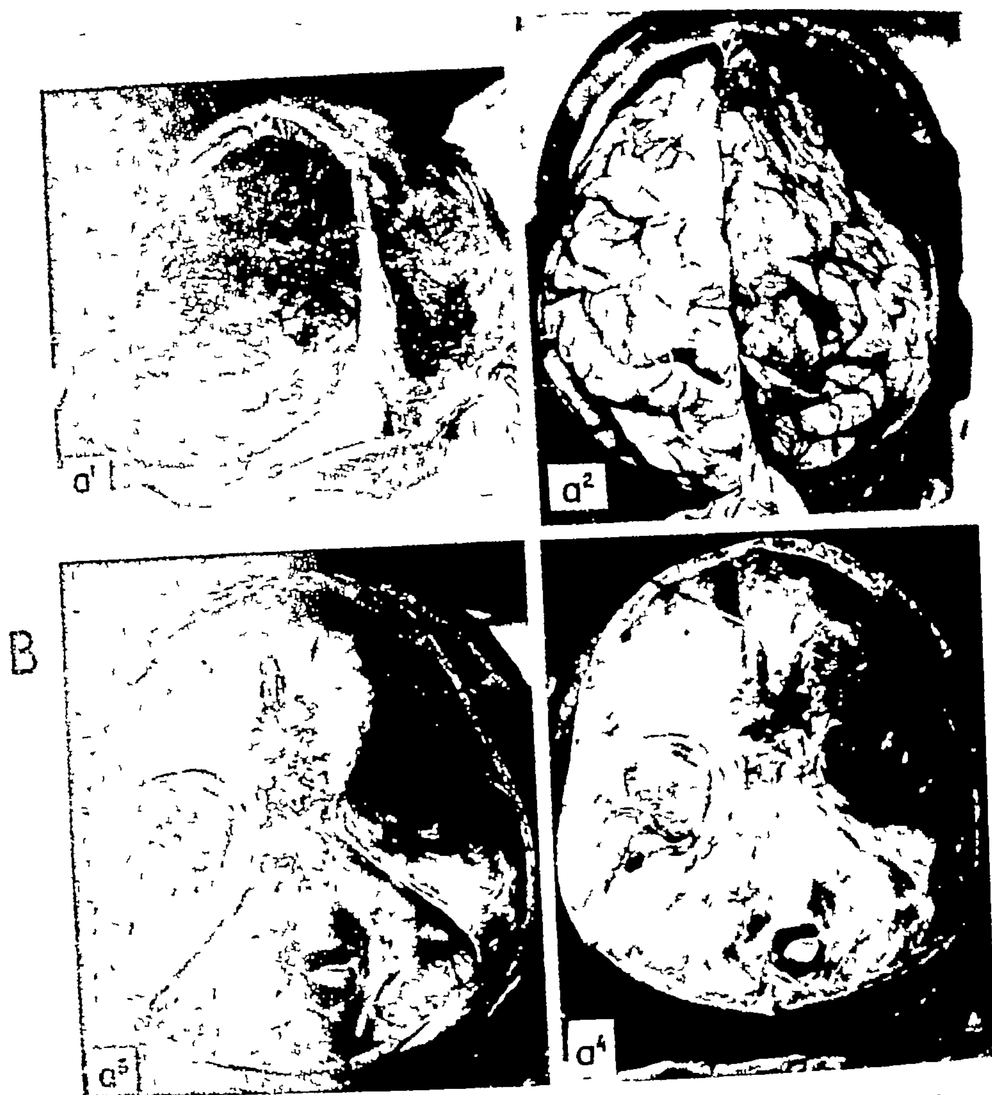


Fig 55 (continued) (B) Oculomotor paralysis with hematoma (a^1–a^5), fronto-parietotemporal hematoma (a^1) extending into middle fossa found on autopsy, cerebral compression (a^2), base of skull with brain removed (a^3), separation of dura from bone by hematoma (a^4), including superior orbital fissure where nerve was compressed

fairly thick, a thin, liquefied portion may rest against the periosteum After 30 days or more, the hematoma is partly liquefied; some hematomas consist of thick liver-like clumps surrounded by liquid; others are enclosed in a membrane resembling that of chronic subdural hematoma, with a dark, watery fluid in the center; and still others are partly liquefied but without a definite membrane.[50, 81]

Middle Meningeal Hematoma

The extradural hematoma of middle meningeal origin is usually a lesion of young adult life, but also occurs in infants and children.[12, 29, 84] It is almost always unilateral but there are a number of reports describing bilateral hematomas,[35, 56, 68, 77] and we have encountered a few in our experience. In most cases, the hematoma is a result of bleeding from both the veins and the artery, the veins having thinner walls and therefore being more easily injured.[46] As a rule, the cause is a fracture crossing the meningeal groove, and lacerating the artery and veins lying in it.

Clinical Picture

The classic picture of this hematoma, consisting of a short period of unconsciousness followed by a lucid interval followed in turn by unconsciousness and focal signs, has been recognized for the past 85 years. However, not all cases present this picture. The state of consciousness may show the following variations (*1*) there may be no unconsciousness at any time (*2*) disorientation, semiconsciousness, and a fairly normal state may alternate for days, (*3*) unconsciousness may set in several minutes to days after injury, (*4*) unconsciousness may occur immediately after the injury and deepen into coma without an intervening lucid interval. Any change in the state of consciousness is important in the diagnosis, and must be watched for carefully.

The presence of a lucid interval helps the diagnosis of massive intracranial hematoma, but is not pathognomonic of middle meningeal hematoma. Moreover, now that deceleration injuries in automobile accidents are common there is a greater likelihood of associated intracranial damage so that the unconscious state may not be interrupted by a lucid interval. The length of the lucid interval varies from 15 minutes to several days or even a month.[48, 50, 58, 62, 81] If the interval is very brief or the patient is in a state of acute alcohol intoxication, the interval may not be noticed.[43]

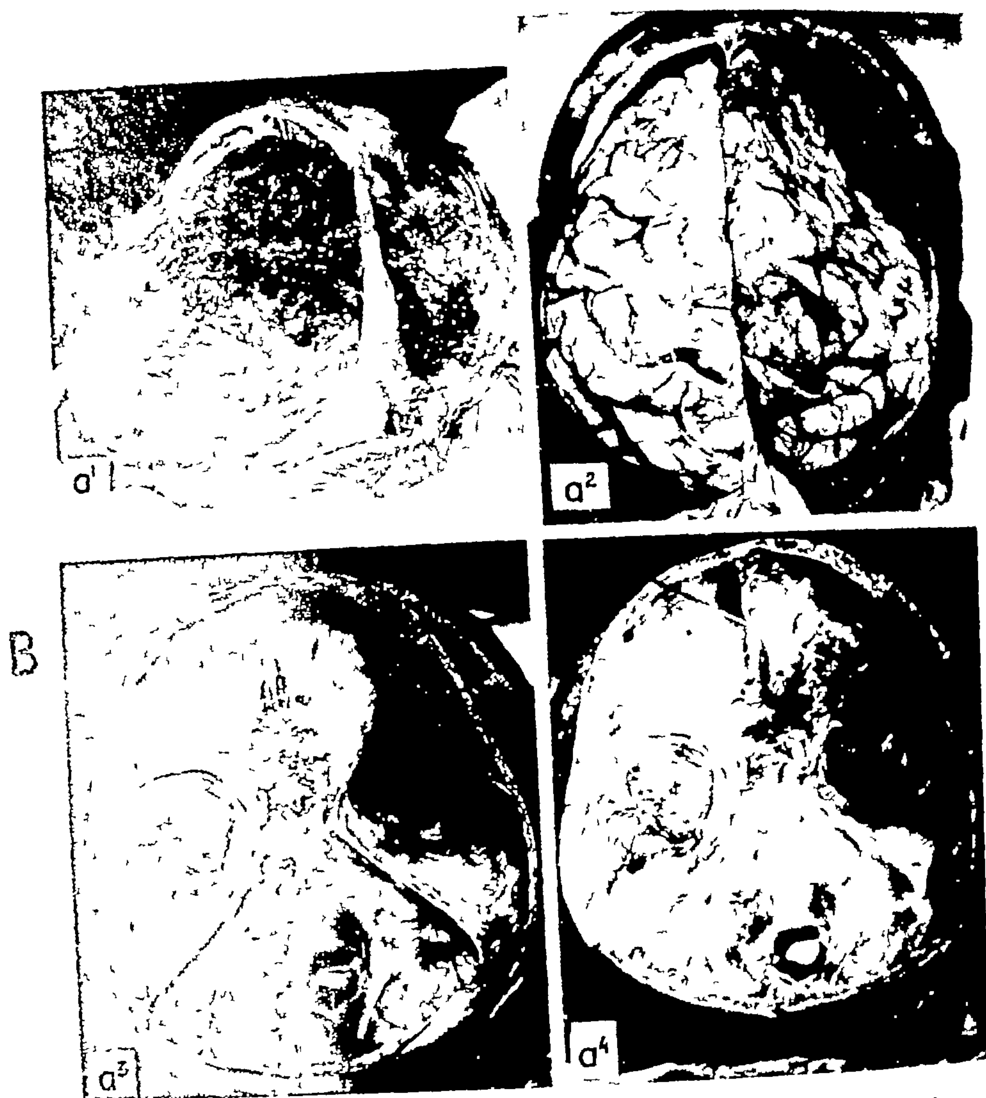


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The *focal signs* in middle meningeal hematoma include ocular abnormalities, motor and sensory changes, and convulsions The ocular abnormalities consist of: (*1*) ipsilateral dilated pupil (Fig 56*A*), an important sign, (*2*) in an occasional case, ipsilateral constricted pupil,[48, 70] (*3*) complete unilateral paralysis of the oculomotor nerve,

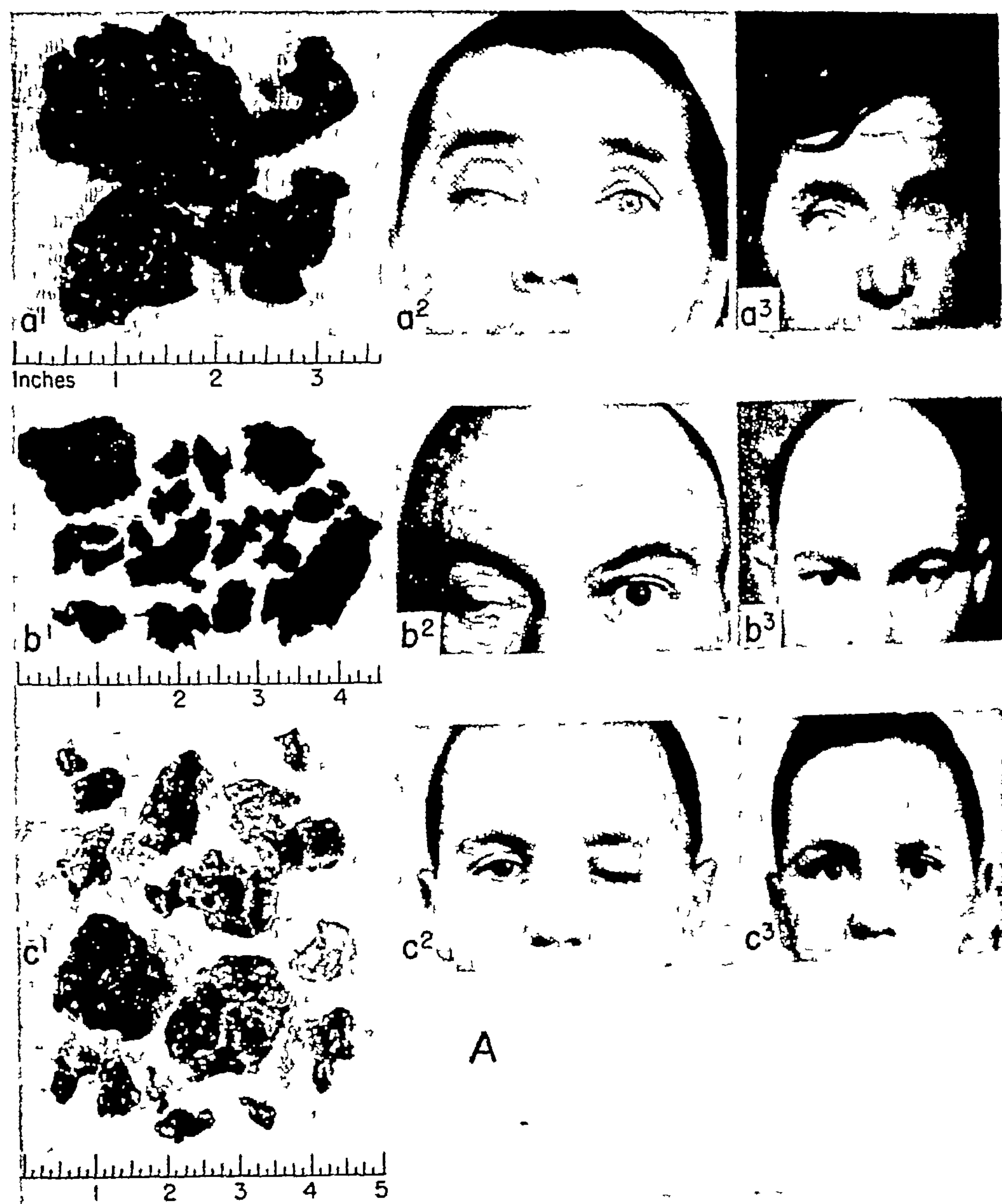


Fig 56 Extradural hematomas of middle meningeal origin (*A*) Hematomas causing oculomotor paralysis, all 3 patients (*a–c*) recovered completely.



Fig. 56 (*continued*) (*B*) Bilateral extradural hematomas (*a*) with linear fracture from temple to temple patient died from cerebral inj r Large extradural hematoma due to depressed skull fracture causing hemiplegia patient recovered after removal of hematoma.

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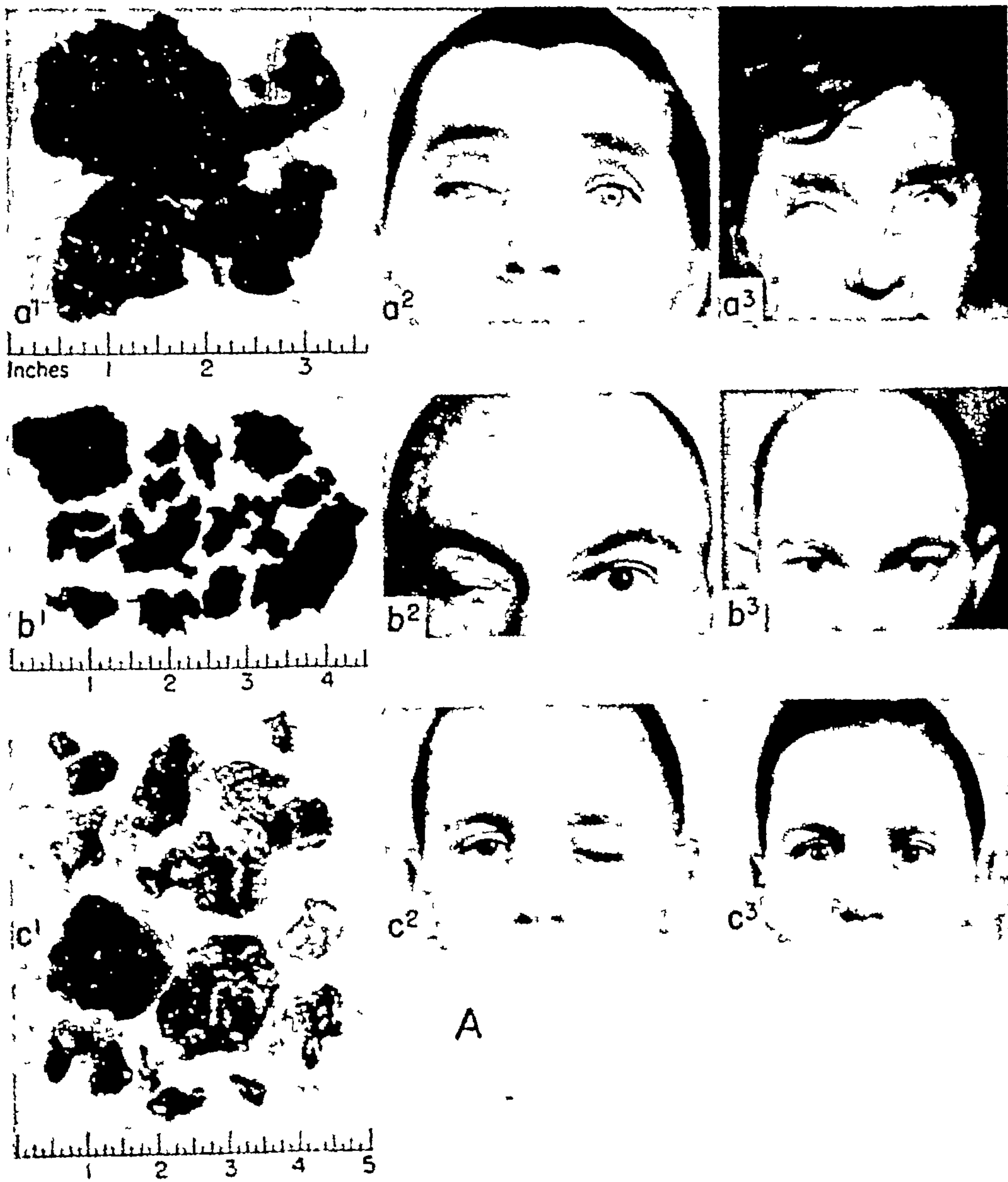


Fig 56 Extradural hematomas of middle meningeal origin. (*A*) Hematomas causing oculomotor paralysis, all 3 patients (*a–c*) recovered completely.

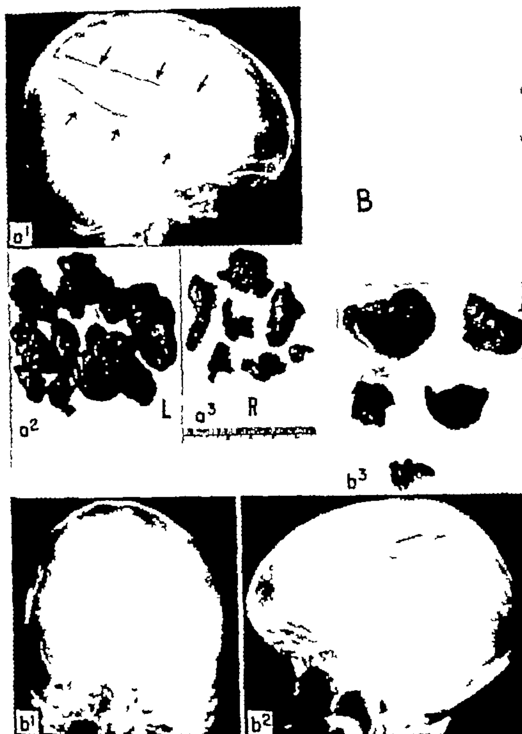


Fig. 56 (*continued*) (*B*) Bilateral extradural hematomas (*a*) with extensive linear fracture from temple to temple patient died from cerebral injuries. (*b*) Large extradural hematoma due to depressed skull fracture causing increasing left hemiplegia patient recovered after removal of hematoma.

The *focal signs* in middle menigeal hematoma include ocular abnormalities, motor and sensory changes, and convulsions. The ocular abnormalities consist of: (*1*) ipsilateral dilated pupil (Fig. 56*A*), an important sign; (2) in an occasional case, ipsilateral constricted pupil;[48, 70] (*3*) complete unilateral paralysis of the oculomotor nerve;

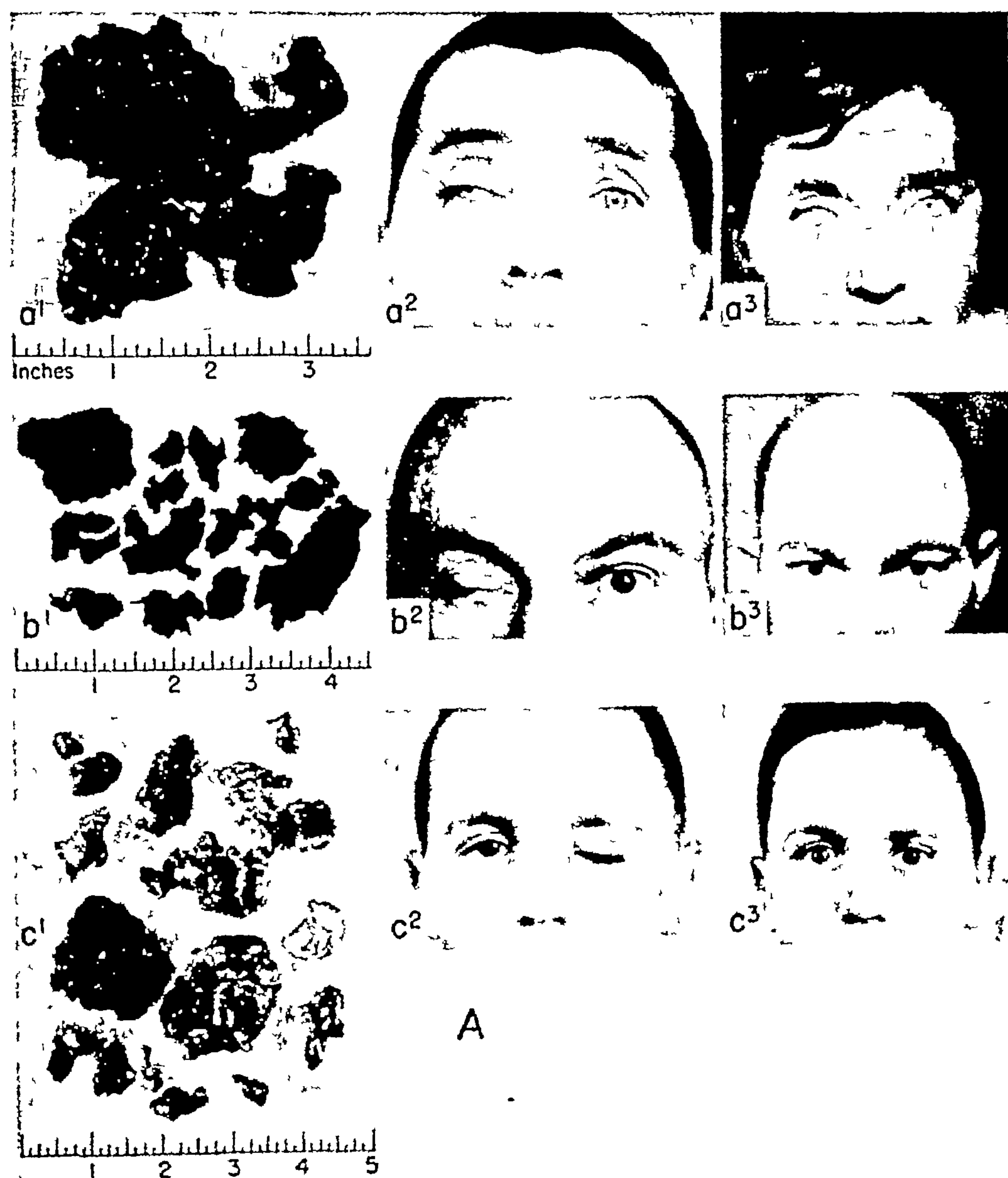


Fig 56 Extradural hematomas of middle meningeal origin (*A*) Hematomas causing oculomotor paralysis, all 3 patients (*a–c*) recovered completely.

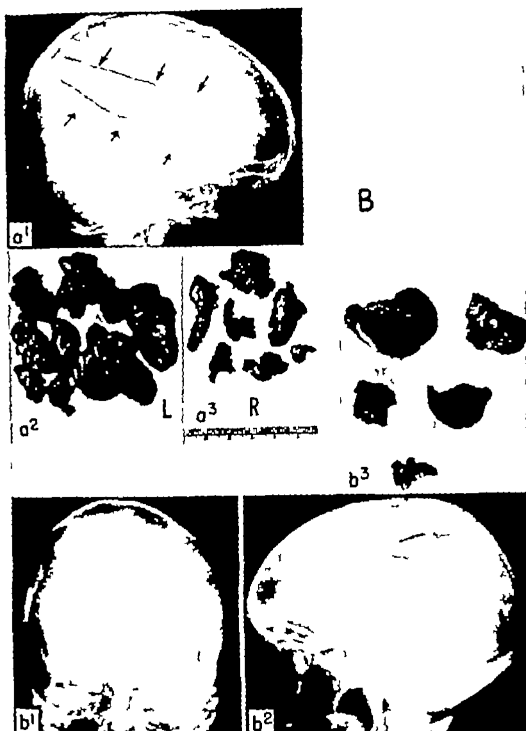


Fig 56 (*continued*) (*B*) Bilateral extradural hematomas (*a*) with extensive linear fracture from temple to temple patient died from cerebral injuries. (*b*) Large extradural hematoma due to depressed skull fracture causing increasing left hemiplegia patient recovered after removal of hematoma.

(4) paralysis of the abducens nerve; (5) trochlear nerve involvement; (6) papilledema or engorged retinal veins; (7) conjugate deviation of the eyes toward the lesion or the side of the hematoma; (8) divergent strabismus. The dilation of the pupil, first noted by Hutchinson,[44] has been variously ascribed to pressure on the oculomotor nerve in the superior orbital fissure,[44] to uncal herniation through the incisura,[86, 90] and to pressure on the nerve by the increased intracranial pressure.[21] If the pressure is not relieved, both pupils may become dilated and fixed. In some cases of hematoma in the parieto-occipital region, the pupils may be of equal size. A dilated pupil, on the other hand, often indicates that the hematoma is in the temporal area extending toward the base.

Among the motor and sensory changes are weakness or paralysis on the contralateral side, aphasia, catatonia, and posturing, so that the patient lies with his face turned toward the side of the lesion. At times, the paralysis may be on the same side as the hematoma.

In some patients, there may be a rigidity involving both sides of the body. It is of uniform character, permitting neither flexion nor extension — a decerebrate or decorticate state without opisthotonos — and is undoubtedly the result of the rapid increase in intracranial pressure. Late in the course of an untreated case, the paresis or paralysis may become bilateral. Bilateral signs with rigidity may be due to uncal herniation, which displaces the brain stem with pressure against the tentorium, multiple foci of cerebral damage, or rare bilateral hematomas.

Occasionally, there may be no localizing signs, either because the hematoma involves the silent portion of the cortex, or because the hematoma has formed as a result of slow seepage, so that the cranial structures have had time to accommodate to the expanding mass.

Convulsive seizures, frequently generalized, may occur. Occasionally, the seizures may be jacksonian, involving one or both sides. The seizures may be the result of pressure upon the motor cortex by the expanding hematoma, or of associated contusions of the motor strip.

The changes in *vital functions* in middle meningeal hematoma in-

clude a slow pulse, full and bounding early in the course of the illness, with a characteristic rate of 40 to 55. Unless treatment is instituted, the rate increases until the pulse is rapid and thready. The respirations are slow and deep, and may become stertorous and of Cheyne-Stokes type. Hypernea and pulmonary edema may occur eventually. In the absence of complications, the temperature is only slightly elevated, ranging from 99 to 101 F. early in the course of the illness. In the untreated case, it may rise considerably. The blood pressure usually remains at normal levels in most cases. In the nontypical case, the vital functions may remain within normal range, or may be markedly deranged, particularly if there is associated severe intracranial damage.

The *cerebrospinal fluid* in most cases of extradural hematoma is bloody, usually because of associated cerebral damage with subarachnoid hemorrhage. This finding, therefore, does not rule out the presence of extradural hematoma. The pressure is usually high, but in some cases it may be rather low. However, a normal or subnormal pressure does not exclude the possibility of extradural hematoma.

The various diagnostic technics described in Chapter IV are used in the diagnosis of extradural hematoma. The roentgenogram almost invariably shows a linear fracture, usually crossing the middle meningeal groove, but in some cases there may be a depressed fracture. However, the absence of a fracture line on the roentgenogram does not exclude the possibility of extradural hematoma, since it may occur without skull fracture or with one so small as not to be visualized on the roentgenogram. In other instances there may be a shift of the pineal gland, an important feature as it may indicate the side of the lesion.

The angiographic features of an extradural hematoma of middle meningeal origin are described in Chapter IV, and illustrated in Figure 29.

Posterior Fossa Hematoma

A tear of the lateral sinus or blood vessels leading into the sinus associated with a fracture or depression may result in an extensive extra

dural hematoma in the posterior fossa These hematomas are usually unilateral, rarely bilateral Many examples of posterior fossa hematomas have been reported in recent years. A low venous pressure (5 to 10 mm Hg) may result in a slow hemorrhage with delayed signs of compression, such as occurred in the case described by Coleman and Thomson [18] Such a hematoma may also result from a perforating wound of the skull and sinus (Fig 57).

In the uncomplicated case, without brain injury, increasing stupor and hypotonia of the extremities may occur [18, 78] Coleman and Thomson noted in their case a lucid interval after a fall from a moving truck, with impact upon the occipital bone; headaches, drowsiness, vomiting, and, later, nuchal rigidity, a generalized hypotonia, arreflexia, and unconsciousness followed Roentgenograms showed a midline occipital fracture extending into the foramen magnum The patient slept on the right side (the same side as the hematoma), whenever his position was changed, he invariably returned to the right-sided position A clot was removed from the right posterior fossa and the patient recovered completely In the majority of cases with such hematomas, the manifestations are not typical and the diagnosis depends on cautious and discerning judgment [14] If there is increasing deterioration of the state of consciousness and lateralizing signs are absent, exploration at or about a fracture line is definitely indicated

Sagittal Sinus Hematoma

Extradural hematoma of superior sagittal (longitudinal) sinus origin may simulate middle meningeal hematoma. The symptoms and signs may be unilateral or bilateral, or first one and then the other. If the lesion remains mainly at the midline, the motor cortex on both sides eventually becomes involved On the other hand, extension of the hemorrhage to one or the other side, with enlarging clot, may produce predominantly unilateral signs and symptoms simulating those of a middle meningeal hematoma The picture may be complicated by asso-

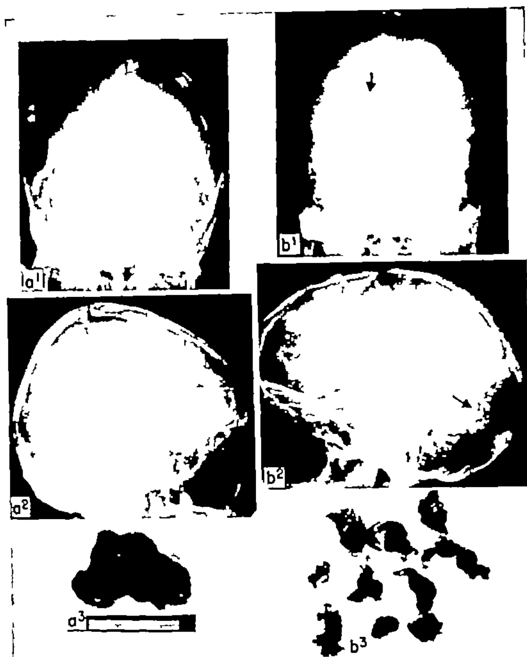


Fig 57 Extradural hematoma due to dural sinus injuries. (*a*) Superior sagittal sinus torn by left interparietal fracture with evulsion of skull triplegia (both lower limbs and right upper limb) signs of bilateral pyramidal tract involvement, and bilateral rigidity (*b*) Lateral sinus torn in perforating injury to right of mid line increasing stupor and signs of bilateral pyramidal tract involvement.

ciated injuries to other parts of the brain and/or subdural and intracerebral hematomas

Tears of this sinus, with thrombosis, may produce the so-called syndrome of the superior longitudinal sinus described by Holmes and Sargent,[10] in which there are bilateral pyramidal tract signs with triplegia or quadriplegia In a case of ours (Fig 57*a*), a huge extradural hematoma at the midline resulting from an evulsion type of fracture was associated with bilateral pyramidal tract signs In another case of sagittal sinus tear with an extensive subdural hematoma, there was marked rigidity of both lower limbs and the left upper limb

Diploic and Emissary Veins Hematomas

Massive hemorrhage from diploic and emissary veins is rare, except possibly in the posterior fossa area There a tear of the emissary vein in the torcular area may result in an extensive hematoma With comminuted, depressed fractures in the mastoid region, the occipital emissary vein may be injured, and extradural bleeding may occur. In 2 of our cases, however, such hematomas did not seem large enough to cause compression Voris[61] has reported large, clinically significant hematomas of diploic vein origin Small, nonsurgical extradural clots from diploic bleeding are frequent concomitants of depressed fractures

Subdural Hematoma[33, 41, 57, 69]

A collection of blood beneath the dura — the subdural hematoma — is a common complication of head injury (Figs. 58-63, *see also* Figs 33, 37-38, 40). It may be acute, subacute, or chronic, depending on its size, rapidity of growth, and the severity and rapid appearance of the signs and symptoms which it produces This is a rather arbitrary classification, and is based on the premise that: (*1*) when bleeding is so severe as to produce signs and symp[illegible] immed[illegible] [illegible]er injury (up to 3 days), the process is [illegible] [illegible]ig is less severe and is tolerated by [illegible]



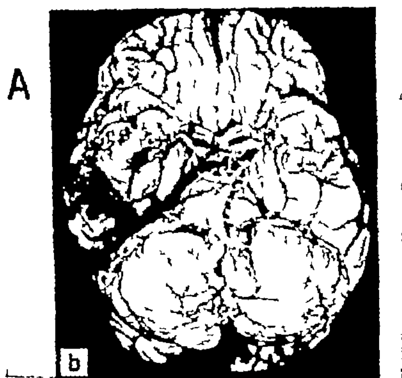


Fig 58. Pathology of subdural hematoma. (*A*) Acute bilateral hematomas (*a*) (*b*) Associated cerebral contusions. (*Continued on next page*)

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A

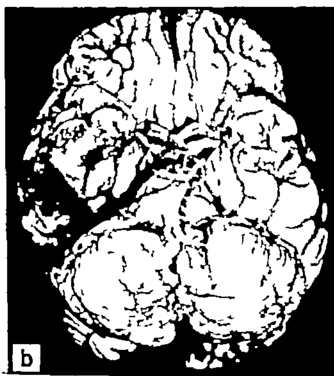


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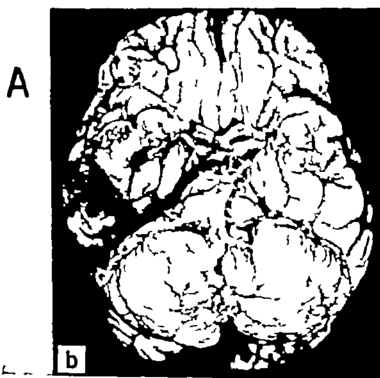


Fig 58. Pathology of subdural hematoma. (*A*) Acute bilateral hematomas (*a*) (*b*) Associated cerebral contusions. (*Continued on next page*)



Fig 58*A* (*continued*) (*c*) Calcified chronic hematoma, found at autopsy, sausage-shaped, 60 Gm mass extended across anteroposterior length of hemisphere, whitish inner lining, solid bone, part of outer lining also calcified, patient had had no symptoms or signs of central nervous system lesion

days), the process is a subacute one, and (3) when the initial hemorrhage is not associated with obvious cerebral trauma and therefore produces no signs for several weeks, or when there is severe cerebral trauma so that the patient is severely ill from the beginning but whose improvement is interrupted by renewed signs of an intracranial dynamic disease, the lesion is chronic (over 16 days)

Chronic subdural hematoma was first recognized in 1657[96] and then, again, in 1747[64] In the early part of the nineteenth century it was thought to be of inflammatory origin[6, 20, 37, 42] Virchow,[91] who subscribed to this theory, described the formation of an underlying membrane and termed it pachymeningitis interna chronica, and the hemorrhage as pachymeningitis hemorrhagica The relation between trauma and chronic subdural hematoma was made clear in 1914[87] and not until 1925[78] was the identity of the pachymeningitis with the chronic subdural hematoma, as we know it, definitely established

Subdural hematomas occur in about 5 per cent of cases of head injury.[10, 66] The mortality rate is highest in the acute hematoma group

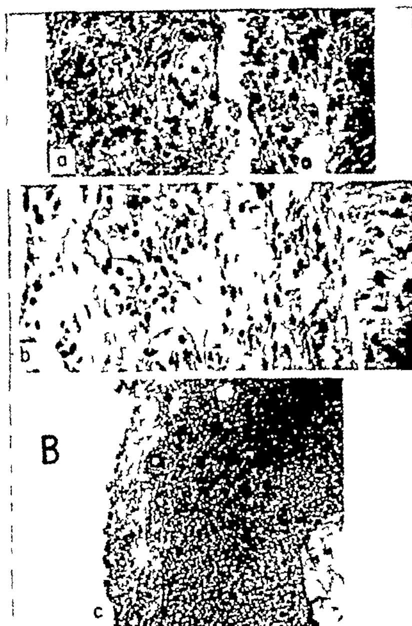


Fig. 58 (*continued*) (*B*) Acute subdural hematoma (*a*) connective tissue proliferation from underside of dura into clot, 18 hours after injury (*b*) Chronic subdural hematoma dura to right, hematoma membrane to left note sinusoidal type of vessels in membrane. (*c*) Chronic subdural hematoma outer wall about 8 mm thick many sinusoidal vascular channels near inner border these tear and bleed, adding to hematoma.

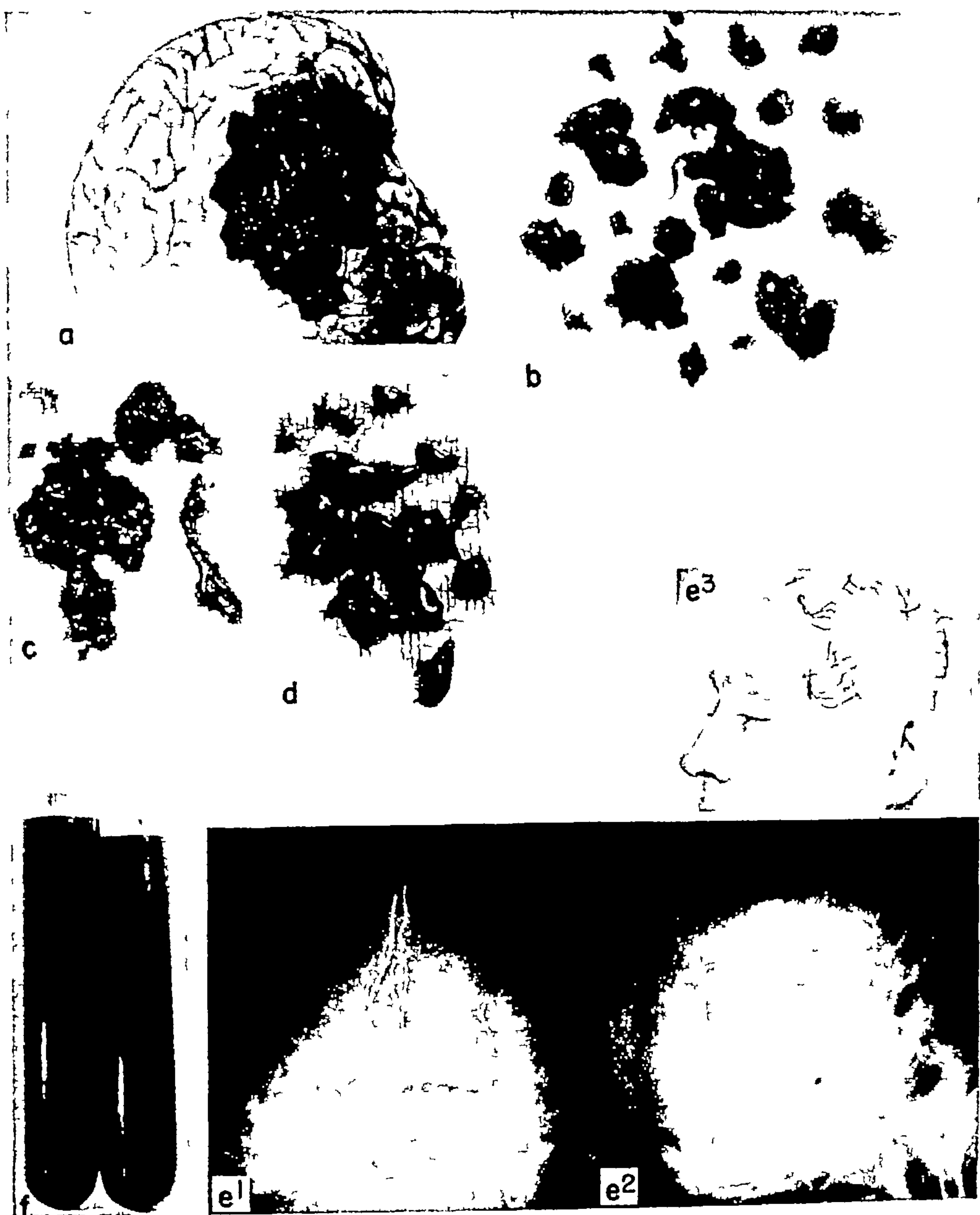


Fig 59 Acute subdural hematoma (*a*) As usually found on autopsy, in association with extensive contusions of frontal and temporal lobes (*b–d*) Hematomas removed through large subtemporal openings or by osteoplastic craniotomy (*e–f*) Hematoma (*f*) removed 9 hours after injury by osteoplastic craniotomy (e^3), angiograms (e^1–e^2) show marked shift of anterior cerebral artery, *see* Figure 38 for EEG.

with the earliest need for surgical intervention.[10, 23, 62] In acute hematoma, the mortality rate ranges from 80 to 50 per cent, in the subacute group it is about 25 per cent, and in the chronic group it is 10 to 15 per cent.

Of 61 patients with subdural hematoma in our recent series of 1,285 cases, 40 underwent operation within 15 days of the injury, 16 within 24 hours or less, 6 within 24 to 72 hours, and 18 within 3 to 15 days. In the first group, 14 died (mortality rate, 81 per cent), in the second group, 3 died (mortality rate, 50 per cent), in the third group, 4 died (mortality rate, 22 per cent) Data on a series of 270 of our cases of subdural hematoma and subdural hygroma are summarized in Table 9.

The subdural hematoma may be unilateral or bilateral,[4, 10] and be located anywhere in the cranial cavity, when bilateral, one hematoma



Fig 60 Subdural hematoma due to tear of lateral sinus by depressed fracture of right occipital area clot pushed posterior portion of hemisphere forward patient at first completely blind this was followed by left homonymous hemianopsia which subsided after 3 months torn sinus repaired by suture, and postoperative skull defect later repaired by tantalum plate (b^3)

TABLE 9 *Data on 270 Cases of Subdural Hematoma and Subdural Hygroma, Surgically Treated**

Data	Number of cases: Hematoma, Acute (65 cases)	Hematoma, Subacute (44 cases)	Hematoma, Chronic (114 cases)	Hygroma (47 cases)
State of consciousness				
Lucid interval	20	29	22	12
Continued unconsciousness or disorientation	32	3	13	5
Headache				
Ipsilateral	0	5	49	7
Contralateral	0	0	0	2
Generalized	0	5	39	5
Condition of pupils				
Dilated, ipsilateral	31	10	22	12
With contralateral hemiparesis or hemiplegia	17	4	22	7
Dilated, contralateral	4	2	7	0
Equal	30	32	85	35
Extraocular palsies				
Cranial nerve III	2	0	9	0
Cranial nerve IV	0	0	1	0
Cranial nerve VI	1	0	1	0
Hemiparesis or hemiplegia				
Contralateral	15	15	27	6
Homolateral	7	4	9	6
Convulsive seizures				
Jacksonian	9	5	8	3
Generalized	7	8	6	4
Cerebrospinal fluid features				
300–650 mm H_2O	9	4	51	0
200–300 " "	18	15	16	8
100–200 " "	—	6	8	15
50–200 " "	7	—	—	—

Bloody	33	22	28	16
Xanthochromic	0	0	22	0
Clear	1	3	25	7
Site				
Left side	28	18	55	19
Right side	26	20	46	15
Bilateral	11	6	13	13
Skull fracture	42	26	33	16
Pineal shift	5	7	18	2
Associated mass lesions				
Extradural hematoma	8	1	0	3
Intracerebral hematoma	6	1	0	0
Subdural abscess	0	0	1	0
Mortality rate, %	44.6	34.1	11.4	29.8

* Recovered 207 cases died, 63 cases. Sex 217 male, 53 female.

is apt to be larger than the other (Fig 58) The site of predilection is over the convexity of the hemisphere, in the frontotemporoparietal area (Fig 59) but hematomas have also been found over the posterior portions of the cerebral hemispheres, over the optic chiasm over the sylvian fissure, and over the cerebellar hemispheres[18] The posterior fossa is a rare site.

Etiology

A tear of the connecting veins between the cerebral surface and the dural sinuses with resulting bleeding, cerebral contusions which tear venous or arterial channels, or extensive intracerebral bleeding with escape of blood to the surface via disruption of cerebral tissue can all cause subdural hematomas. The trauma may occur as a result of blunt head injury without fracture, simple or open depressed fracture, penetrating head injury or fracture, and birth injury The site of the hematoma does not necessarily coincide with the site of the fracture. Occasionally indirect injuries may result in subdural hematoma.[32]

The hematoma over the hemispheral convexity is the result of bleeding from the veins connecting the sagittal sinus and the cerebral surface Bleeding from the parietosphenoid vein results in a temporal hematoma [52] Bleeding from the sagittal sinus itself or from its tributaries may cause the interhemispheral hematoma, and is more commonly seen in penetrating injuries Posterior fossa hematomas result from a tear of the great vein of Galen (which more commonly occurs in birth injuries than in other types of head injury), and tears of the transverse sinuses, cerebellar veins, or the sigmoid sinus and tributary veins [16, 61] Subdural hematoma may occasionally arise from arterial bleeding The association of a ruptured aneurysm[30] and of cerebrovascular disease[34a] with subdural hematoma are examples At times, a subdural hematoma may overlie a brain tumor which has bled In a case of ours, a subdural hematoma and a subdural abscess were found on the same side, a very rare occurrence

Pathology (Fig. 58)

As found on operation, subdural hematomas weigh from 30 to 150 Gm or more A small hematoma which has caused no immediate signs or symptoms may become encysted in the subdural area by a proliferation of connective tissue from the dura, this tissue extends over the borders of the hematoma in a single layer of flat, cuboidal epithelium to its inner aspect, where it lies next to the arachnoid The increased osmotic pressure in the subdural space due to the clot causes diffusion of fluids into the subdural space, thereby increasing the size of the hematoma The center of the hematoma liquefies by lysis, so that the protein content of the cystic mass increases and the osmotic pressure with it [27]

The chronic subdural hematoma, according to Kaump and Love,[47] is the result of a hemorrhage within two layers of the cerebral part of the dura They based their theory on the fact that the hemorrhage is in the parietal area and extends toward the temple, rather than in the middle fossa as might be expected were the blood free to gravitate downward in the subdural space early in the course of the lesion However, the

observation that the brain tends to keep a hemorrhage localized in the subdural space and that the blood does not shift with changes in the head position argues against this concept

Some acute subdural hematomas consist of solid and liquid portions in different areas, for example, a liquid portion in the frontoparietal area and a solid portion in the temporal region (Fig 58) The subacute subdural hematoma is often composed of blood and cerebrospinal fluid, so that most are in a more liquid state than the acute hematomas.

In the chronic hematoma, depending on whether it is liquid or solid there may be a well-developed wall on the dural side and a very thin lining on the arachnoidal side, the latter consisting of a single layer of flat, cuboidal epithelium. In some cases, the hematoma liquefies completely, the liquid gradually growing thinner until it is almost colorless and of high protein content.[23, 44] Some chronic hematomas which are well tolerated for many years solidify within the dural membrane (Fig 61*A*) Calcifications may occur in the clot, in the envelopmg membrane, or in both, and in some hematomas there is actual bone formation (Fig 58) Since von Rokitansky[60] first noted such calcifications, they have been seen repeatedly and visualized on roentgenograms.[9, 12, 19, 21, 28, 43] Particularly in children, small subdural hemorrhages may organize and calcify in later life to be visualized on roentgenograms.

Clinical Picture

The diagnostic features of subdural hematomas are discussed in detail in Chapters IV and V Acute subdural hematoma may be difficult to diagnose in some cases because of the presence of associated cerebral injuries. Unconsciousness lasting for 2 days or more, in the absence of localizing signs, calls for a detailed neurologic examination and the use of all indicated diagnostic technics. In summary the signs and symptoms of acute hematoma are (*1*) Unconsciousness from the beginning or a rapid deterioration of the state of consciousness. (*2*) Abnormal ocular manifestations, including dilatation and occasionally constriction, of a pupil on the side of the hematoma, extraocular paral



Fig 61 Chronic subdural hematoma (*A*) Solid hematoma (*a*), pneumoencephalogram (a^1), hematoma after removal (a^2) and *in situ* (a^3–a^4), note thick outer membrane next to dura and thinner membrane next to arachnoid, outline of osteoplastic craniotomy (a^5), and postoperative pneumoencephalogram (a^6), note visualization of both ventricles and evidence that brain has begun to return to its normal position on left.

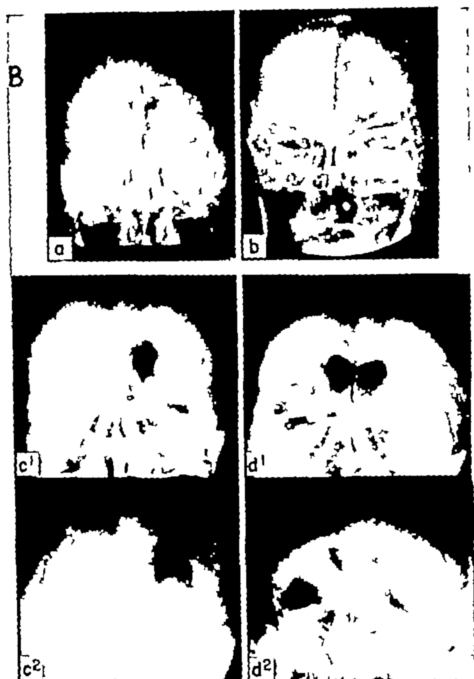


Fig 61 (*continued*) (*B*) Pneumoencephalography (*a*) Right subarachnoid spaces obliterated, but no definite shift of ventricular spaces or third ventricle 100 cc fluid clot found. (*b*) Right subarachnoid spaces obliterated ventricles not visualized large hematoma found on right side. (*c–d*) Pre and postoperative pneumoencephalograms note appearance of ventricle previously compressed, and normal position of ventricular spaces, which are somewhat dilated.

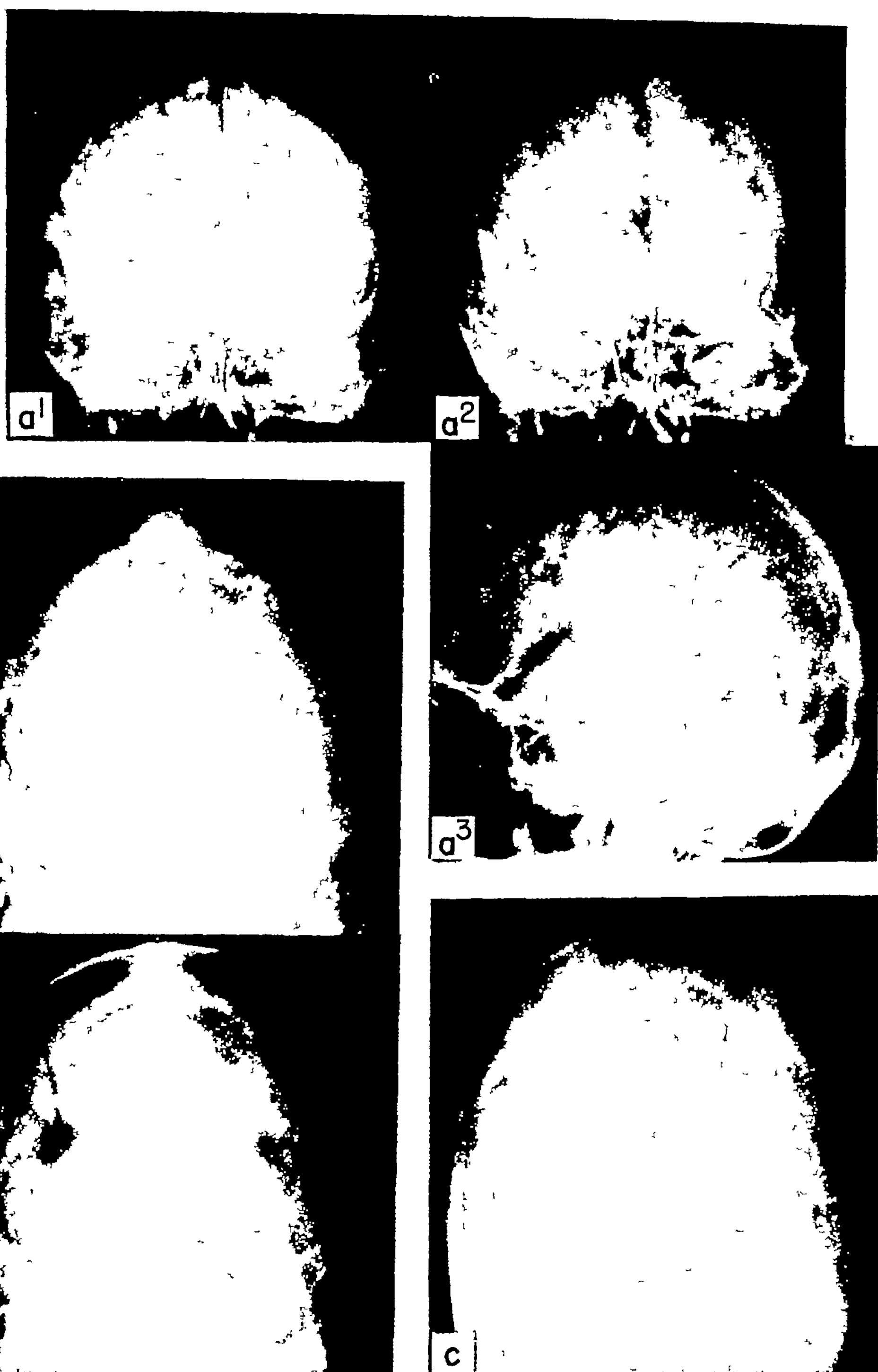


Fig 62 Chronic subdural hematoma (*A*) Pneumoencephalograms (*a*) in a case of bilateral hematomas, left hemisphere compressed, revealing convolutional markings in anteroposterior view, suggesting lesion on left side, but bilateral hematomas found on operation (*b*) Shift of pineal gland and return to normal position after operation (*c*) Angiogram suggested presence of mass on left side, but operation revealed bilateral subdural hematomas and a right extradural hematoma.

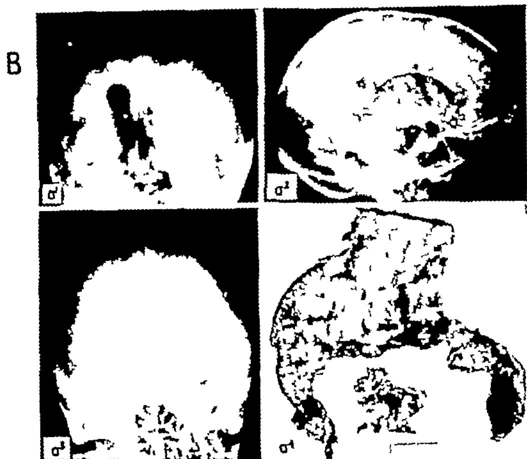


Fig 62 (*continued*) (*B*) Almost completely solid hematoma, 3 months old (*a*¹) removed by osteoplastic craniotomy note shift of ventricular spaces to right and depression of left lateral ventricle, suggesting presence of mass in posteroparietal and occipital areas note also air trapped immediately under hematoma (*a*³) *Continued on next page*)

yses, retinal hemorrhages, and papilledema. (3) Contralateral hemiparesis or hemiplegia with confirmatory pyramidal tract signs (4) Loss of or poor control of half the body or lower facial weakness which may be noted after several minutes of observation or by gentle stimulation. (5) Pyramidal tract signs in the absence of complete paralysis, such as a positive Babinski s sign, unilateral change in deep tendon reflexes, or loss of the abdominal reflex on one side. (6) Catatonic like states with some left-sided hematomas. (7) Convulsions, jacksonian or generalized,

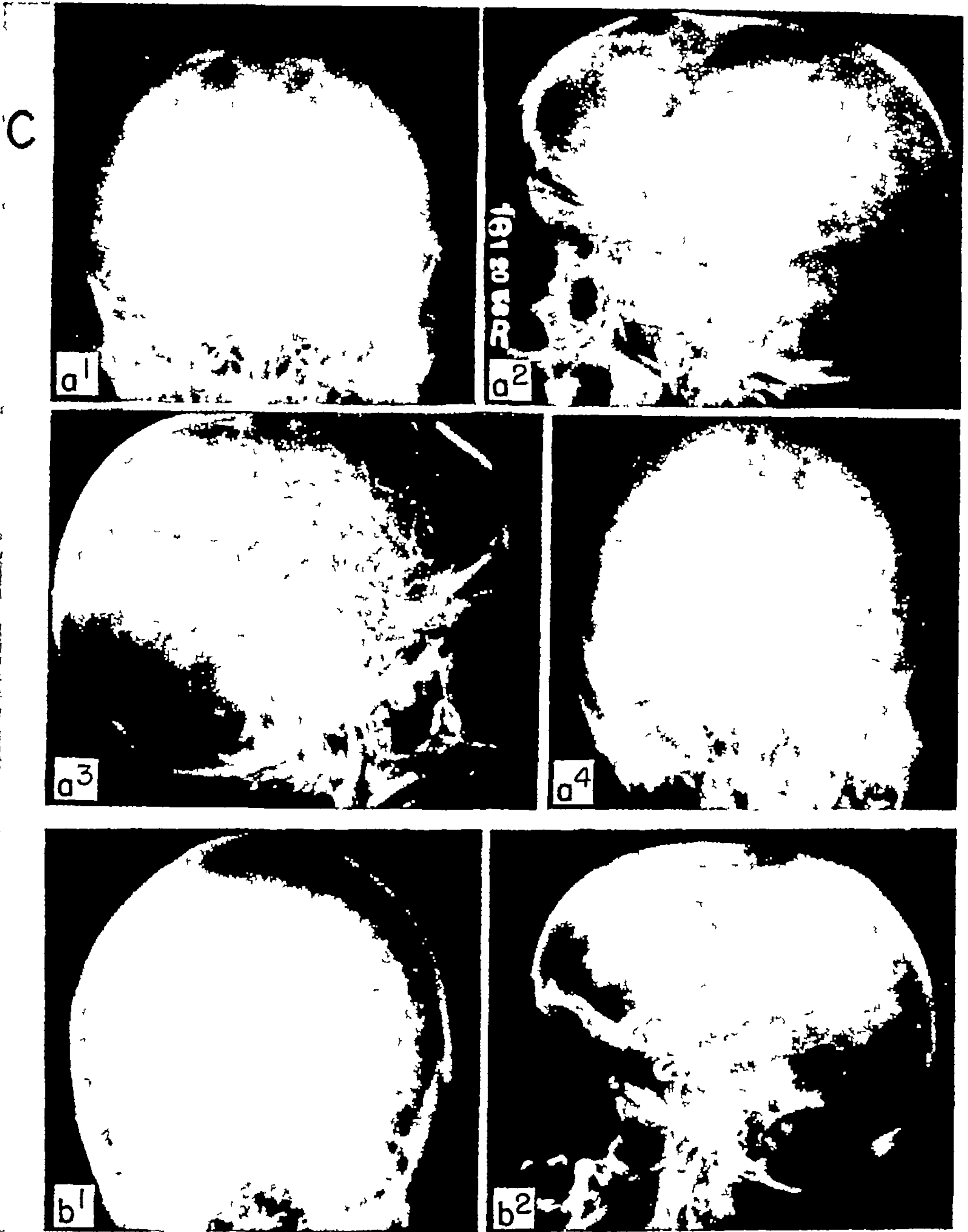


Fig 62 (*continued*) (*C*) Angiography in subdural hematoma (*a*) Bilateral chronic hematomas (right, 40 cc, left 80 cc), anterior cerebral artery bowed to right (a^4) (*b*) Subacute hematoma after a fall, angiogram revealed aneurysm of left cerebral artery, fall probably due to rupture of aneurysm

and decerebrate rigidity (8) Homolateral or contralateral or no fractures.

In the subacute subdural hematoma the unconsciousness or drowsiness with disorientation may last for a few days before further deterioration sets in Other symptoms and signs are much the same as for the acute type.

For the diagnosis of a chronic subdural hematoma all diagnostic measures must be drawn upon if its presence is suspected. Except when there is severe head injury, consciousness is usually normal up to the time of hospital admission The patient with a severe head injury and a developing chronic hematoma continues to improve for 3 to 4 weeks, after which he complains increasingly of headache A semiconscious, stuporous, or unconscious state may then set in In the absence of a severe head injury headache is the most common initial symptom in some cases there is a pronounced personality disorder — nervousness, excitability, or explosive behavior — so that a neurosis may be diagnosed Formerly, an impressive number of chronic hematomas were found in patients in mental institutions and at autopsy This is much rarer now that patients are more scrupulously evaluated before they are committed to an institution

In a large number of cases the headache is over the site of the lesion, and in almost as large a number it is generalized, only occasionally is there a contralateral headache. After lumbar puncture, the headache may disappear for 7 to 10 days Other treatment, too, may affect the character or persistence of the headache

Focal signs may be absent, but sometimes neurologic study may reveal visual field defects, such as a homonymous hemianopsia due to an uncal compression of one or the other posterior cerebral artery, a contralateral hemiparesis or hemiplegia, unequal size of pupils, or an oculomotor paralysis. The hemiparesis is ipsilateral in about 10 per cent of cases, a paradoxic finding explained by contralateral compression of the brain stem against the incisura by uncal herniation. In some 35 per cent of cases, there is contralateral hemiparesis or hemiplegia

Vital Functions

In the acute hematoma, the pulse may be fast and thready early in the course, or sometimes a somewhat slower pulse than normal The respiration may change from slower than normal to hyperpnea, in some cases becoming stertorous and of the Cheyne-Stokes type The blood pressure may rise in some A fever of 101 to 103 F. is common

In subacute hematoma, the vital functions are somewhat more stabilized, except as the patient's state begins to deteriorate A slower than normal pulse may then become rapid and thready, respiration which heretofore had been normal may become rapid, hyperpneic and of the Cheyne-Stokes type, the blood pressure may increase

In the chronic form, vital functions are quite normal until advanced cerebral compression occurs Bradycardia may occur, infrequently, the respiratory rate may decrease and the blood pressure increase

Cerebrospinal Fluid

The cerebrospinal fluid is often bloody in patients with acute and subacute hematoma, but the amount of blood present is no indication of the severity of the lesion Subarachnoid hemorrhage may be the cause of the bloody fluid As fluid is allowed to escape, the red color becomes more intense, indicating the presence of greater amounts of blood at higher levels of the cerebrospinal axis The pressure is usually high, 300 to 450 mm of water, but may be low in some cases A low pressure and a high blood content is a serious prognostic sign However, normal or subnormal pressures do not rule out the presence of massive unilateral or bilateral hematomas

In some cases of chronic hematoma, the fluid is bloody or xanthochromic, and the pressure is usually elevated (200 to 300 mm or more of water), occasionally, it is between 200 and 100 mm A clear cerebrospinal fluid is found much more often in chronic than in acute and subacute hematoma Lumbar punctures are well tolerated Some patients improve temporarily after the removal of cerebrospinal fluid.

The *diagnosis* of subdural hematoma is established by the history physical examination, and symptoms and signs (*see* Chapter V), and with the aid of all the diagnostic technics described in Chapter IV which may be necessary The features of subdural hematoma disclosed by the various technics are also to be found in Chapter IV

Prognosis

In acute subdural hematoma the prognosis is usually poor, and the more acute the manifestations the poorer the prognosis. The convalescence of many who survive is prolonged Posttraumatic epilepsy as well as other severe neurologic deficits and personality disorders may develop in some cases. Improvement is more rapid and complete in patients up to the age of 17, and unexpected recovery with relatively few or no neurologic deficits may occur

In subacute hematoma, much better results are obtained with treatment than in acute hematoma. Good recovery may be expected in about 50 per cent of the cases.

Treatment is successful in about 90 per cent of the cases of chronic hematoma and recovery after removal of the clot may be rapid Posttraumatic epilepsy occurs in less than 10 per cent of the cases, in our experience, and mental deficits are uncommon. In the presence of cerebral injury, however, the prognosis depends on the severity of the associated injury

Intraparenchymatous Hemorrhages Intracerebral Intracerebellar, and Petechial[2, 11]

Intraparenchymatous hemorrhages may vary from massive collections to petechial or punctate lesions. Hemorrhages up to about 5 mm in diameter are considered petechial. Larger lesions are usually designated as hematomas and may have surgical significance Although pathologically all intraparenchymatous hemorrhages are similar, the clinical manifestations and principles of management are not the same

Petechial Hemorrhages (see Fig. 24)

Petechiae are commonly found on autopsy. In about 45 per cent of cases that come to autopsy, multiple petechiae up to 5 mm in diameter, but usually about 1 mm , are found in various parts of the nervous system It is of interest that in a series of 133 consecutively autopsied cases (*see* Table 12) there were 54 instances of petechial hemorrhages, of these, 25 were in the brain stem, and all but 6 of the cases were dead on admission to the hospital, none of the 6 survived longer than 36 hours

Petechiae are small, nonsurgical hemorrhages which may occur either as a result of stresses due to absorption of energy at the time of impact, or as secondary manifestations of herniations, torsions, and shifting of intracranial structures causing kinking or compression of blood vessels Petechiae may also be caused by embolism, particularly fat embolism Primary traumatic petechiae usually occur along the path of the forces acting upon the tissues Thus, small petechiae are common in the brain stem, medulla, pons, posterior thalamus, striatum, and even the spinal cord With the creation of pressure gradients in the cranial cavity as a result of blows upon the head, there may be tension (tearing apart), compression (squashing), or cavitation, causing tears in tissues and bleeding with resultant petechiae

Petechiae can also be secondary manfestations of ischemia, which gives rise to increased permeability and diapedesis and transudation of blood into the tissues Thus, associated with uncal herniation, there may be small hemorrhages in the posterior thalamus and in the brain stem The so-called sentinel petechiae seen about the border of hematomas and deep contusions are due to compression of vessels in the vicinity of the contusion or hematoma, resulting in increased permeability and hemorrhages by diapedesis

Small petechiae are probably absorbed, so that at autopsy later in life no evidence of their presence is found [74] Larger clots (5 to 10 mm. or larger) are transformed into cystic cavities filled with clear fluid or with masses of glial fibers forming bands of tissue crossing the cavity, and

after absorption of the clot, with phagocytes and plasma cells. The cystic cavity is surrounded by a rather thick fibroglial wall. The microscopic characteristics of the cysts, whether small or large, are essentially the same. Years later, secondary hemorrhage may occur if a blood vessel crossing a larger cyst is disrupted

The symptoms and signs of petechial hemorrhage depend on the location the number, and the size of the petechiae. Decerebrate states, akinetic mutism, dyskinesias, bilateral pyramidal tract signs varying degrees of violence and restlessness, hemiplegia, or triplegia may be seen with petechiae in the brain stem subfrontal areas, and the basal ganglions (*see* Chapter V)

Intracerebral and Intracerebellar Hematomas

According to Echlin, intraparenchymatous hemorrhages of massive size occur in about 1 per cent of patients with head injury We have found an incidence of 1 6 per cent.

Large intraparenchymatous hematomas usually occur in the frontal and temporal portions of the brain (Fig 63) A few are seen in the parieto-occipital area, and the cerebellar lobes are involved occasionally The temporal and frontal localization is accounted for (*1*) by the stresses in these regions as a result of mass movements of the intra cranial structures at the time of impact (2) by pressure gradients with below normal pressure resulting in cavitation and tearing of tissues, and (3) by the anatomic relationship of the frontal and temporal poles with the lesser wing of the sphenoid and the roof of the orbit so that deep contusions or tears occur easily with resultant intracerebral hema tomas. The hematomas are frequently on the same side as a fracture, but occasionally may be bilateral. Table 10 summarizes the data on a series of 38 cases.

Hematomas may also be found in association with penetrating and perforating wounds. In penetrating wounds, the hematoma may form in the path of the bullet or shell fragment (Fig 63) The incidence of intracerebral hematomas in the Korean War was high Low velocity

TABLE 10 *Data on 38 Cases of Intracerebral Hematoma**

Data	*Number of cases*
State of consciousness	
Lucid interval	15
Continued unconsciousness or semiconsciousness	11
Headache	
Generalized	10
Contralateral	1
Condition of pupils	
Dilated right	3
Dilated left	2
Side involved	
Right	18
Left	20
Papilledema	7
Hemiparesis	
Ipsilateral	2
Contralateral	20
Cerebrospinal fluid features	
Pressure > 500 mm H_2O	5
" 400 " "	4
" 300 " "	10
" 200 " "	7
" 100 " "	3
Bloody	21
Xanthochromic	7
Clear	1
Convulsive seizures, generalized	1
Focal signs	22
Site	
Frontal	6
Temporal	26
Parietal	3

Frontal and temporal	1
Intracerebellar	2
Skull fracture	22
Pineal shift	2
Type of surgery	
Osteoplastic craniotomy	7
Trephine	31

* Recovered, 17 cases died, 21 cases mortality rate, 55.2 per cent. Sex 30 male 6 female.

penetrating objects may also tear vessels and be associated with large intraparenchymatous hematomas (*see* Fig 50)

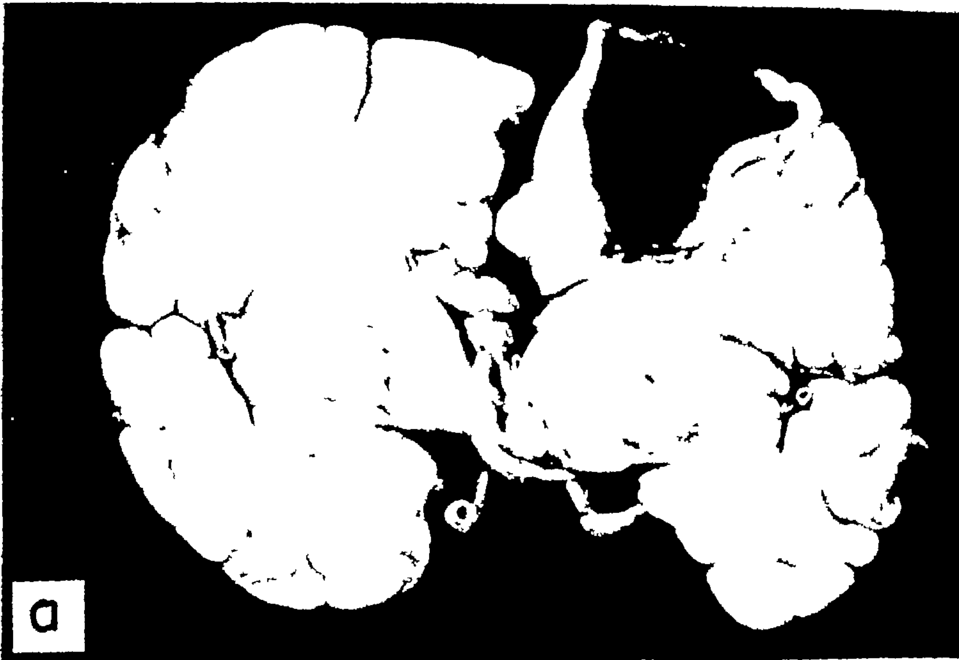
Pathology

Larger hematomas which do not cause signs of compression may at first liquefy by hemolysis, and eventually consist of a liquid center with some clotted material about the periphery or toward the cyst wall The fluid gradually thins and pales, and finally may become clear When the cyst communicates with the ventricles or the subarachnoid channels, as it does in some cases the so-called porencephalic cyst is formed Calcification of intracerebral hematomas has been reported [21]

Clinical Picture

The symptoms and signs depend on the location of the lesion In the absence of severe concussion there may be a lucid interval with increasing headaches. Occasionally, jacksonian or generalized convulsions may be seen. An intracerebral hematoma in the temporal lobe may cause a *contralateral* paresis of the upper limb and face which may progress to a complete hemiparesis or hemiplegia accompanied by stupor and coma If the left temporal lobe is *involved, speech disturbances* also may be present In patients with associated signs of severe concussion there may be coma from the beginning The hemiparesis or hemiplegia may then be found several hours to several days after

A





ıt temporal

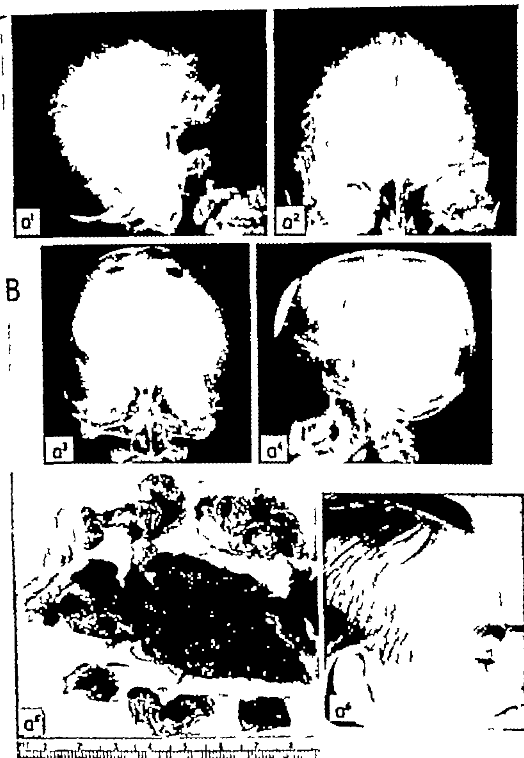


Fig. 63 (*continued*) (*B*) Hematoma due to penetrating bullet injury angiograms 4 days after primary debridement (a^1–a^2) show evidence of mass lesion in right frontal lobe bullet still in cranial cavity increasing stupor and pyramidal tract signs bilaterally large hematoma (a^5) removed and pericallosal branch of anterior cerebral artery ligated tantalum cranioplasty (a^3–a^4) 7 months later for skull defect, and patient in profile (a^6)

the initial injury Occasionally, there may be a hemiplegia on the same side as the hematoma

In some patients with deep temporal or occipital hematomas, there may be an associated homonymous hemianopsia, either found before operation or after the patient improves enough to cooperate in a visual field examination

Pupillary inequality with a dilated pupil on the side of the lesion is not as frequent a finding as in subdural and extradural hematomas Papilledema is uncommon About one-third of the patients do not have a skull fracture Occasionally, the fracture is on the side opposite the hematoma A shift of the pineal gland is seen infrequently

Intracerebellar hematomas, particularly when they are subacute or chronic, are difficult to diagnose In war injuries, roentgenographic evidence of a bullet, or bone or shell fragments in the posterior fossa suggest the presence of the hematoma The following case histories illustrate the manifestations of intracerebellar hematomas Severe psychotic manifestations may be seen with intracerebellar lesions

Case 1 The patient was a 27 year old woman without a history of unconsciousness at the time of injury On hospital admission, 10 days after injury, she was in a disturbed but drowsy state, this had been progressing during the preceding 3 days No skull fracture could be found on roentgenograms Localizing signs suggesting a hematoma were absent Mild nuchal rigidity and a slight fever were present A tentative diagnosis of meningitis was made But when the cerebrospinal fluid was found to contain 40 red cells and 75 white cells per cubic millimeter, mainly lymphocytes, an encephalitis seemed more likely The next day she became irrational and died in a few minutes Autopsy disclosed a partly liquid, partly solid, clot the size of a hen's egg in the right cerebellar lobe

The history and the marked mental abnormalities in this case are difficult to explain on the basis of the cerebellar involvement alone However, the increased intracranial pressure due to obstruction of the cerebrospinal fluid pathways by the clot may have caused the psychotic manifestations

Case 2.[*] The patient, a soldier in World War II had been injured 9 days earlier There was an insignificant appearing wound in the neck, to the left of the midline. The patient complained of severe headache there was vomiting a horizontal nystagmus on left lateral gaze, and incoordination in the left upper and lower extremities. Roentgenography revealed a comminuted fracture of the occipital bone to the left of the midline. At operation, a perforation into the posterior fossa was found, with cerebellar tissue extruding through it. Incision of the dura was followed by the sudden extrusion of about 40 cc. of dark clotted blood after which the herniated cerebellum receded and brain pulsations were observed The wound healed by primary intention in spite of the delayed debridement. Two years later the patient was well except for some disability in the left hand.

Case 3 Some 2½ months after a head injury the patient began to complain of severe headache, and papilledema appeared. Mild right cerebellar signs were observed with ataxia and adiadokokinesis of the right upper limb. A lesion in the posterior fossa was diagnosed by means of ventriculography On operation a huge cyst filled with straw-colored liquid was found in the right cerebellar lobe. The cyst wall tissue contained hemosiderin and old blood It was thought that the cyst was a result of an intracerebellar hematoma which had become absorbed, and that the residual cyst had so compressed the fluid pathways as to cause an increase in intracranial pressure

The last case is an example of a subacute or chronic intraparenchymatous cerebellar mass lesion. Hemorrhage into a tumor may produce the same picture as a subacute or chronic intracerebellar hematoma The diagnosis of this form of intracerebellar hematoma therefore cannot be absolutely established unless the tissues can be studied microscopically and unmistakable evidence of an old hemorrhage is found and the patient recovers satisfactorily and permanently after operation.

The *diagnosis* of intracerebral hematoma in the frontal, temporal, and parietotemporal regions is best confirmed by angiography ventriculography or diagnostic trephination An intracerebellar hematoma may be suspected in the presence of a perforation in the occipital area over the cerebellum in penetrating injuries or in the presence of a linear skull fracture involving the occipital squama in a

patient who is markedly disturbed and whose condition is deteriorating In such a case, exploration in the vicinity of the fracture line is undoubtedly indicated Electroencephalography may be of value in some cases Schneider and associates[84] have located some intracerebellar hematomas by looking for them along the lines of force as deciphered from contusions, lacerations, and fractures

The cerebrospinal fluid is bloody or xanthochromic in most cases of intracerebral hematoma, and the pressure is often elevated, ranging between 500 and 300 mm of water, although in some cases the pressure is between 200 and 100 mm Occasionally, the fluid may be clear

As for the *prognosis*, patients who survive usually do so with an impaired level of mentation, and many have long-lasting personality disorders Posttraumatic epilepsy occurs in about 20 per cent of cases

Subdural Hygroma[17, 57, 72]

The subdural collection of cerebrospinal fluid which occurs in many cases of head injury is known as the subdural hygroma or hydroma It is interesting to note that Sir Percivall Pott[76] recognized this condition, that it was then described in 1896[60] and again in 1916[75] Some consider it as a type of subdural hematoma,[70] but often this would seem to be an unjustified conclusion The collection may or may not be associated with an extradural or a subdural hematoma Large collections have been found in the posterior fossa under the tentorium in patients whose tentorium was incised to decompress the brain stem for uncal herniation[70]

The cause of subdural hygroma has been variously given as (*1*) the presence of small amounts of blood in the subdural space, with a resultant increase in osmotic pressure and migration of fluids to the area, (*2*) a tear in the arachnoid, (*3*) cerebral contusions, with resultant edema and increase in osmotic pressure, (*4*) inflammatory disease of the arachnoid, (*5*) physicochemical changes following severe head injury, and (*6*) blast injuries[1]

The quantity of fluid may be large or small, and clear, straw-colored or xanthochromic. A wet brain and a considerable subdural collection of fluid are occasionally found in a case that comes to autopsy

The correct diagnosis is usually made at operation The signs and symptoms mimic those of subdural hematoma. In the infant a subdural tap may establish the diagnosis, and injection of a small amount of air may reveal the extent of the involved area In about 75 per cent of cases, the cerebrospinal fluid is bloody, and the pressure is usually below 300 mm of water

After evacuation of a large collection of subdural fluid, air may accumulate in the subdural space (*see* Fig. 30)

In our 1952 1954 series of 1,285 cases, there were 7 cases of extradural hematoma, 61 of subdural hematoma, 16 of intracerebral hematoma, and 3 of subdural hygroma. The incidence of hemorrhagic mass lesions in this series was 6.7 per cent.

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Chapter VIII

COMPLICATIONS OF HEAD INJURY

Aside from the immediate and late effects of head injury, various complications can sooner or later develop. Some are exceedingly rare, others more or less common. Among the rare complications are diabetes mellitus, diabetes insipidus, Simmonds' disease, gastrointestinal hemorrhage, and gastrointestinal ulceration. More common are pneumocephalus, otitis media, mastoiditis, osteomyelitis, meningitis, intracranial suppuration, and cerebral fat embolism.

Pneumocephalus[5, 6, 10, 16, 19]

A collection of air in the cranial cavity (Fig. 64) is a frequent result of a craniomasal or cranioaural fistula. Occasionally, air enters the cranial cavity via an open fracture of the vault. Usually, the complication develops a few days after injury,[14] but cases have been reported in which it occurred weeks or years after the injury.[2, 11, 12] The site of the pneumocephalus may be extracranial, subperiosteal, extradural, subdural, subarachnoid, intraventricular, or intracerebral.[14] The intracerebral aerocele is probably the result of the air being forced in,

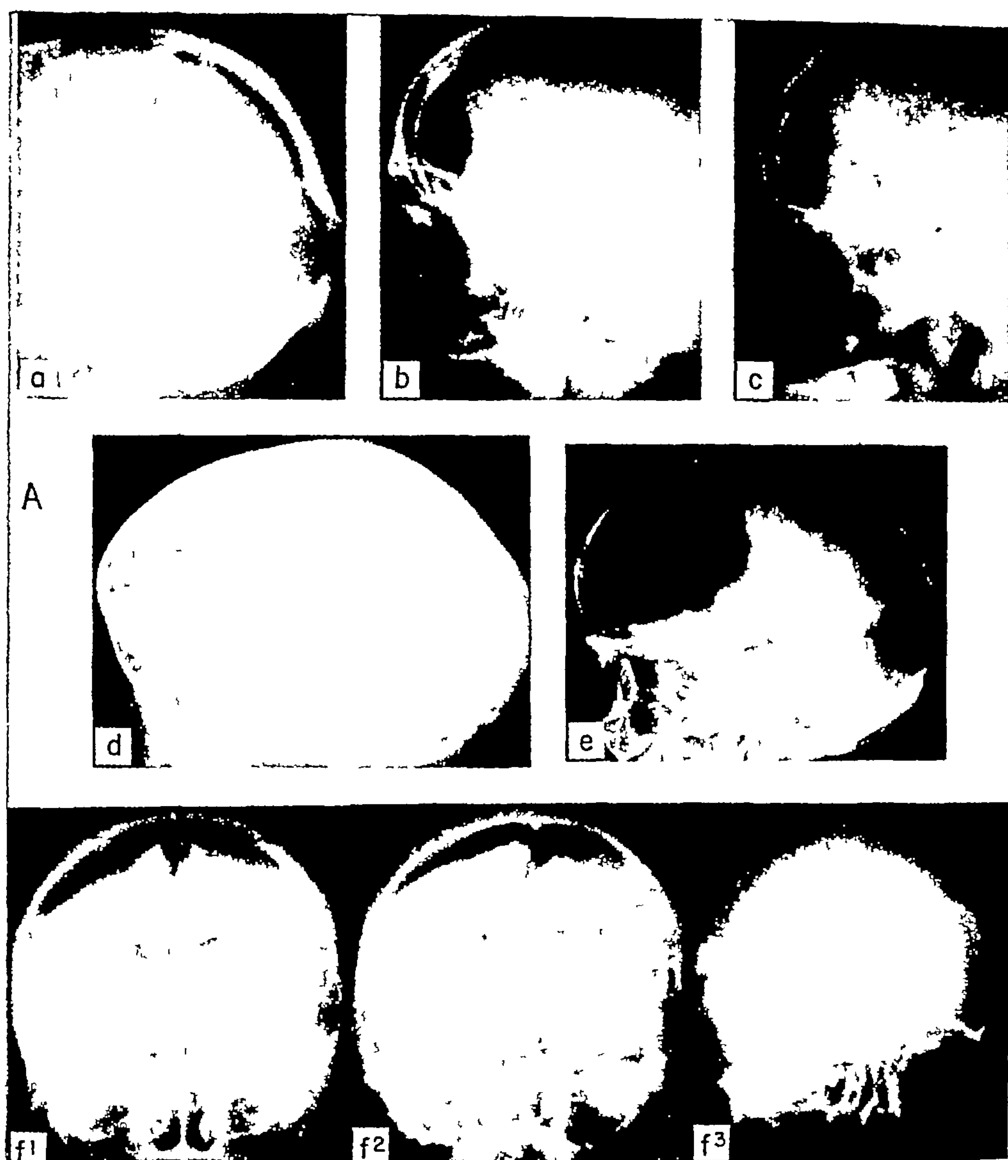


Fig 64 Pneumocephalus (*A*) Subdural pneumocephalus (*a, c*) due to cranionasal fistula with cerebrospinal fluid rhinorrhea (*b*) Subdural pneumocephalus due to cranioaural fistula with cerebrospinal fluid otorrhea (*d*) Pneumoventriculum after open fracture of occipitoparietal junction at midline (*e*) Pneumatocele which caused extensive destruction of frontal lobe (*f*) Progressive disappearance of air from subdural area and from ventricles in a case of cerebrospinal fluid otorrhea

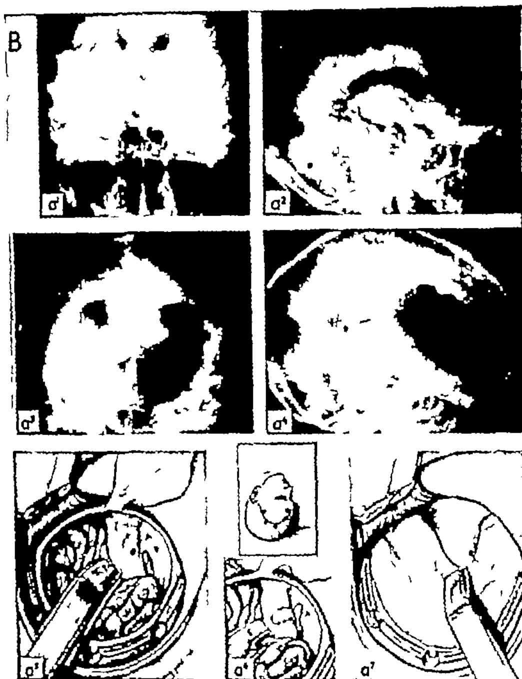


Fig. 64 (*continued*) (*B*) Mechanism of pneumatocele formation. (a^1–a^2) Fracture of left frontal bone extending into sinus. (a^3) Roentgenogram 11 weeks later shows large cavity in left frontal lobe patient admitted to hospital with meningitis. (a^4) After intraspinal air injection relation of pneumatocele to ventricular system (a^5–a^7) Operative repair of craniomasal fistula involving cribriform plate to left of midline by means of reflected falx cerebri.

through a cranionasal communication with adhesions between the brain, the piarachnoid, and the dura, by sneezing, coughing, or nose blowing. The air dissects a progressively enlarging area in the brain, particularly if its escape is prevented by a ball-valve action in the fistulous tract Eventually, cerebral tissue is destroyed and replaced by a cystic cavity The process may take a relatively short time· 11 weeks in the case illustrated in Figure 64*B*.

Roentgenography establishes the diagnosis The clinical features are severe headache (intense in acute pneumocephalus, accompanied by a shocklike state) With subdural, subarachnoid, and intracerebral pneumocephalus, there may be focal phenomena, hemiparesis, hemiplegia, aphasia, convulsive seizures, and deterioration in the conscious state In the patient who is already unconscious because of the initial severe effects of the head injury, some of the focal phenomena may not be evaluated accurately But in patients with a chronic cranionasal or cranioaural fistula the onset of pneumocephalus may be ushered in with severe headaches and focal phenomena, these usually subside after a few days of bed rest

A summary of the findings in 14 of our cases of traumatic pneumocephalus is given in Table 11

TABLE 11. *Data on 14 Cases of Traumatic Pneumocephalus*

Data	*Number of cases*	*Deaths*
Cause		
Cranionasal fistula with cerebrospinal fluid rhinorrhea	8	
Cranioaural fistula with cerebrospinal fluid otorrhea	5	
Open occipital fracture	1	
Treatment		
Surgical	9	1
Conservative	5	1

Otitis Media and Mastoiditis

Otitis media and mastoiditis are now uncommon complications of head injury Before the days of antibiotics and the sulfonamides, they were seen fairly frequently Although otitis media may be present a few days after an acute head injury characteristically it occurs 4 to 6 weeks after trauma. Continuing mucopurulent discharge from the middle ear and roentgenographic evidence of involvement of the mastoid air cells are the usual signs of mastoiditis.

Osteomyelitis of the Skull

This complication of head injury, the result of inadequate treatment of an open fracture, has now grown rare. According to Browder, osteomyelitis of the skull may be classified as (*1*) Pott s puffy tumor, (2) osteomyelitis due to drying of the skull in the absence of a covering scalp (*3*) osteomyelitis due to improperly treated open skull fracture, and (*4*) osteomyelitis associated with middle ear and accessory nasal sinus fractures.

Pott s puffy tumor, a swelling of the scalp over an osteomyelitic area, is rarely seen now Occasionally, a puffy area occurs in association with a retrograde thrombophlebitis from infection of the cavernous sinus or other focus of infection In such cases, there may also be one or more brain abscesses.

Extensive destruction of the scalp may expose the skull and thus allow the bone to dry out But proper treatment should prevent the occurrence of osteomyelitis from this cause.

Osteomyelitis due to an improperly treated open skull fracture is seen occasionally Such a wound may continue to drain for many weeks, the skin becoming soft and boggy because of the associated cellulitis. Exposure of such an area may reveal the presence of hair, dirt and other

particles, or fragments of bone with some purulent matter in the extradural area Thickening of the dura, resulting from the organism's attempt to limit progress of the infection, may occur In chronic osteomyelitis, there may be destruction of bone and repair and osteitis, with thickening and sclerosis of bone. Only occasionally does an osteomyelitis due to trauma progress to invade uninvolved portions of the skull The bone may be soft, and is easily excised by the rongeur In some cases, the involved bone may be completely decalcified

Osteomyelitis of the mastoid area and the bones around the paranasal sinuses may occur in patients with fracture in these areas or with persisting patent cranioaural or cranionasal fistulas Prompt treatment of such fistulous tracts is the best method of preventing an osteomyelitis in these regions Such bone infection of the skull may be associated with extradural or subdural abscesses

The diagnosis of traumatic osteomyelitis is made on the basis of (*1*) the presence of roentgenographic evidence of bone destruction and sequestration, (2) the presence of chronic inflammatory scalp involvement after an open skull fracture, and (*3*) a history of trauma in a patient with known sinus disease, in whom retrograde thrombophlebitis and diploic involvement may infect the skull (Fig 65).

Meningitis

Meningitis, 5 to 8 days after injury and occasionally sooner, used to be a more common complication of head injury than it is now The sulfonamides and antibiotics are responsible for this reduction in incidence Onset is usually fulminating, with severe headaches, disorientation, and coma In some cases, the first sign may be a convulsive seizure High fever, nuchal rigidity, and delirium are common The cerebrospinal fluid is turbid, the polymorphonuclear cell count is high, and fresh smears at times reveal the presence of the infective organisms

Meningitis may occur as a late complication of a temporal bone fracture or a fracture of the anterior fossa involving the cribriform plate,

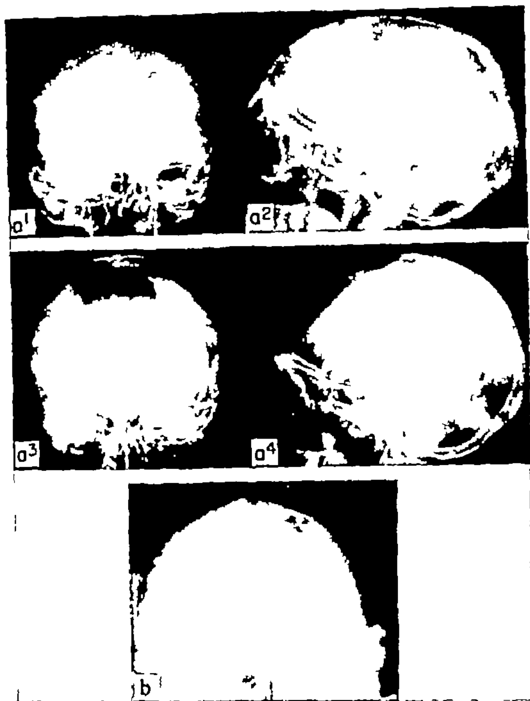


Fig 65 Osteomyelitis of skull subdural abscess, and brain abscess. (*a*) Osteomyelitis of frontal bone and intracranial abscesses after blow on forehead in presence of sinusitis· suppuration of scalp and subcutaneous tissues drained in doctor's office· patient later admitted to hospital with convulsions, right-sided paresis, and aphasia about 85 cc. of pus escaped when dura was opened symptoms recurred 5 weeks later and left temporofrontal intracerebral abscess was found eventual cure followed repeated drainage and penicillin instillation. (*b*) Osteomyelitis of skull after frontal blow

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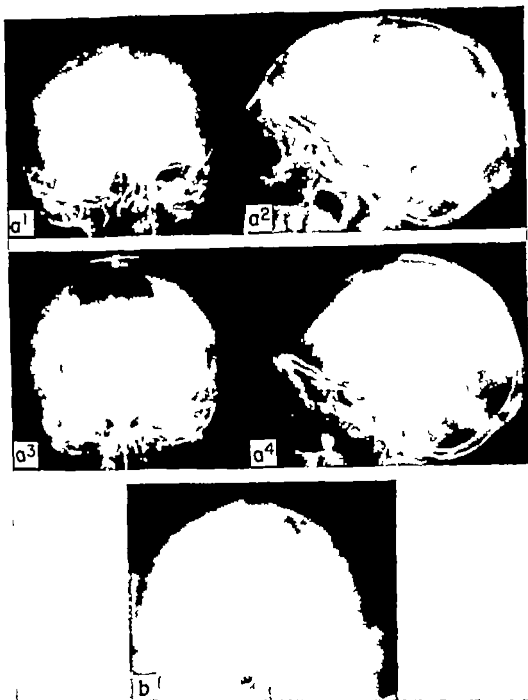


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with the formation of a patent cranionasal fistula. Repeated attacks of meningitis may occur in such cases

Intracranial Abscess

A collection of purulent material or abscess may develop after head injury of varying severity anywhere in the cranium: beneath or in the scalp, extradurally, subdurally, in the brain, or in the ventricles.

Etiology

The most common causes are direct extension of infection from an untreated or incompletely debrided open skull fracture or wide spread of infectious material by a penetrating object, particularly high-velocity missiles A retrograde thrombophlebitis and perivascularitis resulting from a scalp and/or skull infection is another cause of intracranial suppuration Still another cause is the spread of infection along preformed pathways, such as the prolongation of the subarachnoid spaces about the blood vessels and nerves, the internal ear, the internal acoustic meatus, and the endolymphatic duct

Particularly in closed head injury, the possibility of blood-borne infection lodging within the head and resulting in intracranial suppuration should be kept in mind. Infection of the ear after a head injury may be followed by a temporal or cerebellar abscess as a result of extension of the infection through perivascular pathways, retr ade thrombophlebitis or through preformed The adoxic brain abscess results from the pa hery to the left side of the heart th the cardiac chambers, so that infecti o the brain without passing through

Foreign bod lded in the l found to be purulent c around a b blade, and tumor, in years af

minor blow to the frontal area, in the presence of active or quiescent sinusitis, may be the means of activating the infection, with spread to the brain and abscess formation (Figs 66A, 67) A deep cerebral contusion may become the site of a chronic brain abscess, the infection being

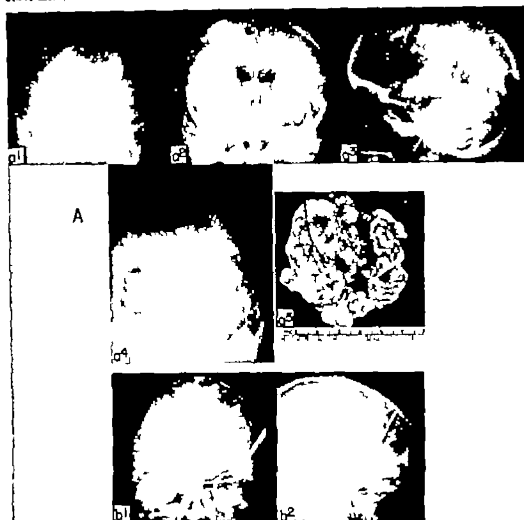


Fig 66. Brain abscess. (A) Ten years after penetrating injury by shotgun in back of head and neck (a^1) lead pellets in right cerebellar fossa (a^2) internal hydrocephalus and probable mass in posterior fossa (a^2–a^3) postoperative roentgenogram and mass removed from left cerebellar lobe (a^4–a^5) (b) Roentgenograms showing broken-off stiletto in left frontal lobe 2 weeks after injury for which patient had been given first-aid treatment elsewhere patient was admitted to hospital with complaints of headaches, some papilledema, and cerebrospinal fluid pleocytosis exploration revealed large abscess around blade abscess was excised completely site irrigated with penicillin solution, wound closed without drainage penicillin injected twice, and wound healed by primary intention. (*Continued on next page*)

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Foreign bodies embedded in the brain substance have repeatedly been found to be the focus of a purulent collection,[8, 9] for example, an abscess around a broken-off knife blade, and a walled-off abscess, simulating a tumor, in the cerebellum 11 years after a bullet injury (Fig *66B*) A

minor blow to the frontal area, in the presence of active or quiescent sinusitis, may be the means of activating the infection, with spread to the brain and abscess formation (Figs. 66*A*, 67) A deep cerebral contusion may become the site of a chronic brain abscess, the infection being

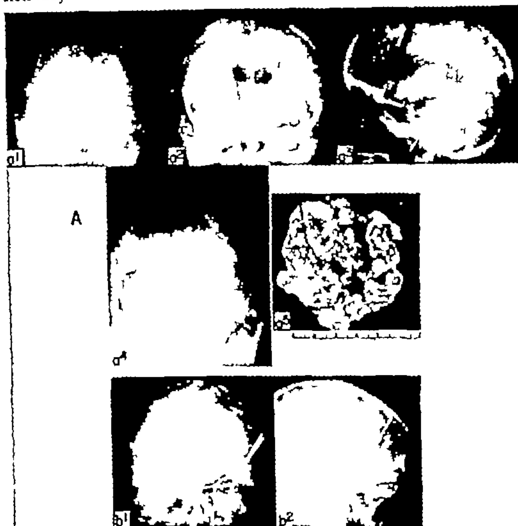


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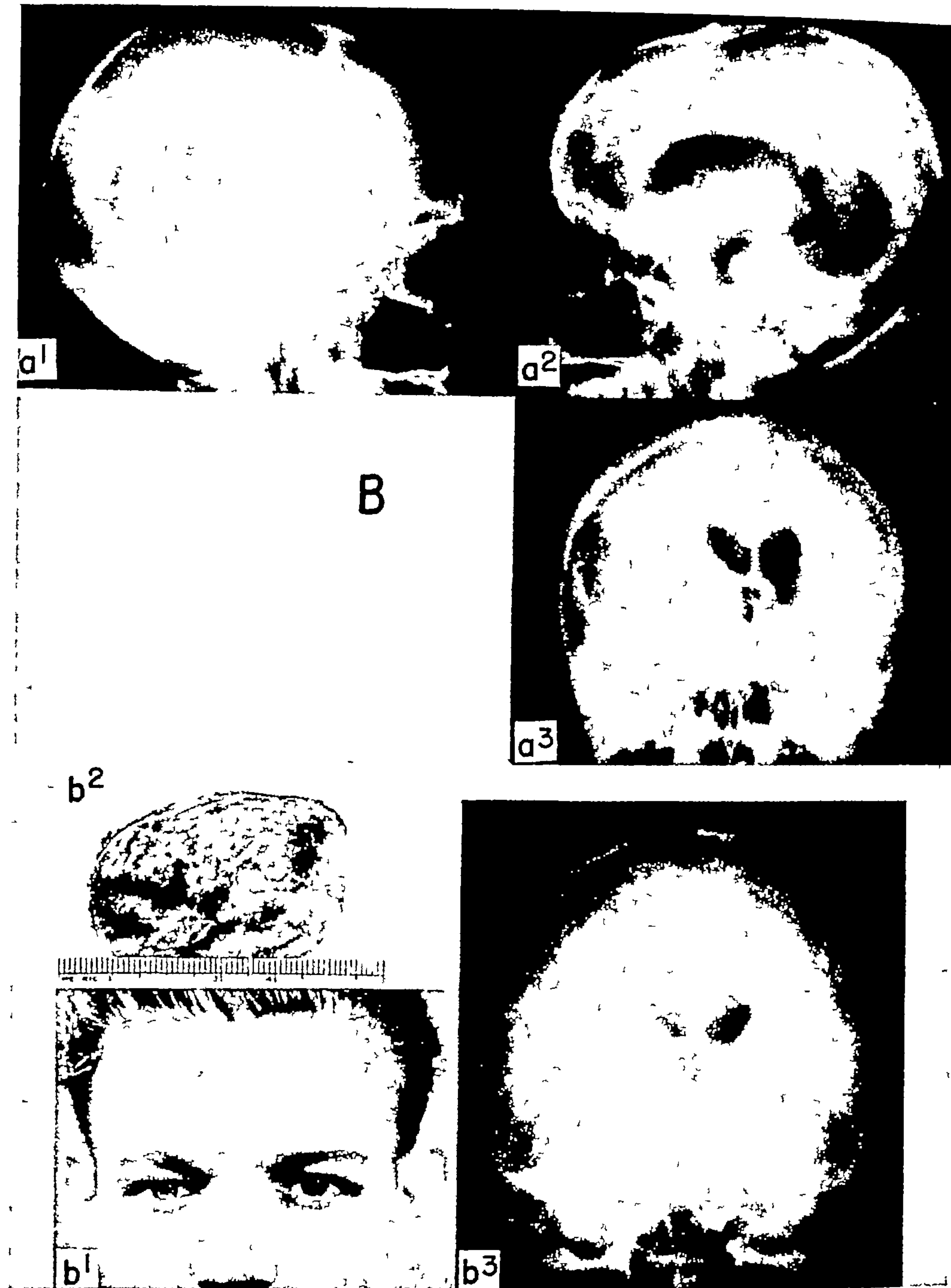


Fig 66 (*continued*). (*B*) Abscess after open depressed skull fracture, treated elsewhere (*a*), preoperative roentgenogram and pneumoencephalograms (a^1–a^3) showed mass lesion in right occipital area, secondary debridement of old wound, and removal of considerable necrotic cerebral tissue, purulent matter, and several bone fragments, together with complete excision of involved area, local application of penicillin, and closure of wound without drainage resulted in healing by primary intention (*b*) Encapsulated abscess 3 months after blow in right frontal region (b^2), patient 1 year after excision of abscess (b^1), and preoperative pneumoencephalogram (b^3) showing mass in right frontal lobe



Fig 67 Osteomyelitis of skull and brain abscess 2 months after incomplete debridement of compound skull fracture. (a^1–a^2) Pre and postoperative roentgenograms amount of osteomyelitic bone excised (a^3–a^4) presence of abscess established by pneumoencephalography area drained and Thorotrast injected to visualize abscess cavity (a^5–a^7) exploration of area revealed 2 discrete abscesses (a^8)

caused by organisms arriving via the blood stream. Any surgical procedures on the head may be followed by intracranial abscess formation, although this is now a rare cause. However, the antibiotics now in common use occasionally suppress infection only temporarily, the infection soon begins to progress, resulting in extensive suppuration. Postoperative appearance of the characteristic signs of intracranial suppuration should therefore arouse suspicion of its presence.

Pathophysiology

The pathophysiologic features of an intracranial abscess after a closed head injury are essentially the same as those of a nontraumatic abscess. As a rule, the abscess is walled off or encapsulated. The subdural abscess is usually enveloped in a neomembrane, the extradural one walled off by a neomembrane developed on the dura. The neomembrane in subdural abscess may closely resemble that of a chronic subdural hematoma, being thick on the side of the dura and thin on the arachnoid side. In the encapsulated brain abscess there is a suppurating center surrounded by vascularized necrotic tissue, this in turn being bordered by a zone of hyperemia and glial fibrosis, and possibly a zone of gliosis.[4]

The abscess resulting from penetrating injuries, in which there is direct implantation of infectious material, or the cerebritis due to the presence of a foreign body, organic material, or pulping of cerebral tissue, is usually a nonencapsulated one, with extensive tissue necrosis. This may also occur in acute abscess due to blood-borne infection, a spreading encephalitis, with necrotic cerebral tissue enclosed by a wall of acutely inflamed tissue, may be found in such cases, and possibly eventual perforation of the ventricular wall. Traumatic extradural abscess is usually associated with involvement of bone or osteomyelitis.

Ventricular infection or ventriculitis occurs occasionally, one ventricle being more definitely involved than the others. For instance, a lateral ventricle may be filled with turbid fluid due to the presence of pus, yet the contralateral ventricle may have relatively clear fluid.

Symptoms and Signs

In general, the diagnosis of suppuration in the cranial cavity is based on the presence of (1) increasing drowsiness (2) localizing signs (3) low grade fever, (4) a pleocytosis of the cerebrospinal fluid high total protein content, and high pressure, (5) high leukocyte count (6) electroencephalographic evidence of focal involvement and (7) *roentgenographic evidence of bone involvement* There may be focal or generalized convulsive seizures. A history of otitis media may suggest the possible ipsilateral involvement of the temporal lobe or the cerebellum The *various diagnostic technics (roentgenography, pneumoencephalography* ventriculography and angiography) may help to establish the diagnosis by disclosing possible causes for suppuration or the site of the purulent collection, and the possible presence of intracerebral involvement.

A subdural abscess is characterized by the precipitous onset of severe headache, drowsiness, stupor, focal or generalized convulsive seizures, nuchal rigidity, hemiparesis, and focal phenomena Papilledema and signs of meningitis, and, if the lesion is on the left side, aphasia may appear The abscess usually becomes manifest 6 to 10 days after onset of the illness leading to subdural suppuration, such as an upper respiratory infection, sinusitis, or an injury over a potentially or actually infected area.

The diagnosis of brain abscess in war injuries is in some respects easier than that of traumatic brain abscess in civilian practice. Signs of increased intracranial pressure, a fungus cerebri, or the presence of an incompletely debrided wound in a patient with a deteriorating state point to the possibility of a brain abscess or cerebritis The scalp wound indicates the probable site of the intracranial lesion. Roentgenograms of the skull may disclose loss or damage of bone or the presence of metal and bone fragments in the missile's path

We have had 11 cases of traumatic brain abscess in civilian practice, and 33 of cerebral abscess and cerebritis complicating penetrating head injury in war experience. The mortality in the latter was 27 per cent, while among the civilian group, 2 of the 11 died. Figures 65, 66, and 67 illustrate some of our civilian experience.

Fat Embolism

Cerebral fat embolism as a complication of head injury more usually occurs when long bone fracture is associated with the head injury.[26] The reported mortality rate from this complication ranges from 5 per cent[28] to 20 per cent.[24]

Pathophysiology

The bone marrow is the probable source of the fat emboli. It has been shown that fat globules up to 12 μ in diameter can pass through the pulmonary capillaries,[28] that glass spheres considerably larger than the diameter of the pulmonary capillaries pass through the pulmonary vessels of the dog and rabbit,[18] and that there are arteriovenous shunts in the human lung many times the accepted diameter of capillaries which permit passage of glass spheres up to 500 μ.[22] Since the diameter of the cerebral capillary is 8 μ and of the arteriole 20 μ, capillary and precapillary obstruction by fat emboli seems a reasonable assumption.

Fat in the form of tiny particles (chylomicrons) is normally present in the blood, and is therefore assumed to be harmless. It has recently been suggested that fat emboli may be caused by a deranged relation between the chylomicrons and the erythrocytes.[21] Another theory is that fat emboli are caused by the stress of injury which results in increased concentration of blood lipids and lipase and in the disturbed balance of the formed blood elements.[7]

Pathology (Fig 68)

In some cases of cerebral petechiae, microscopic examination of cross sections reveals that the petechiae are thrombosed capillaries or arterioles, with "rings" of necrosis and erythrocytes.[21] These petechiae are more prominent in the white matter than in the gray In other

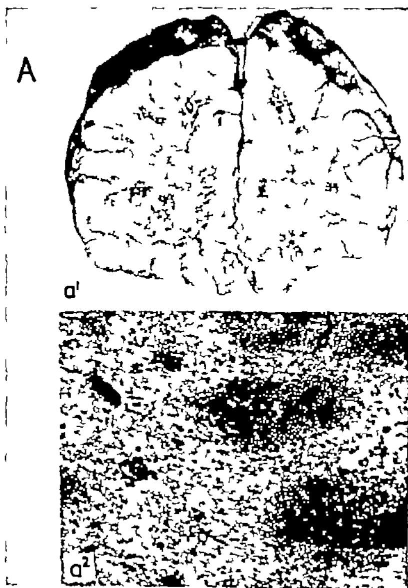


Fig 68. Fat emboli. (*A*) Petechial hemorrhages (a^1) involving white matter almost exclusively fat globules (a^2) in centers of petechiae, revealed by Sudan III stain. (*Continued on next page*)

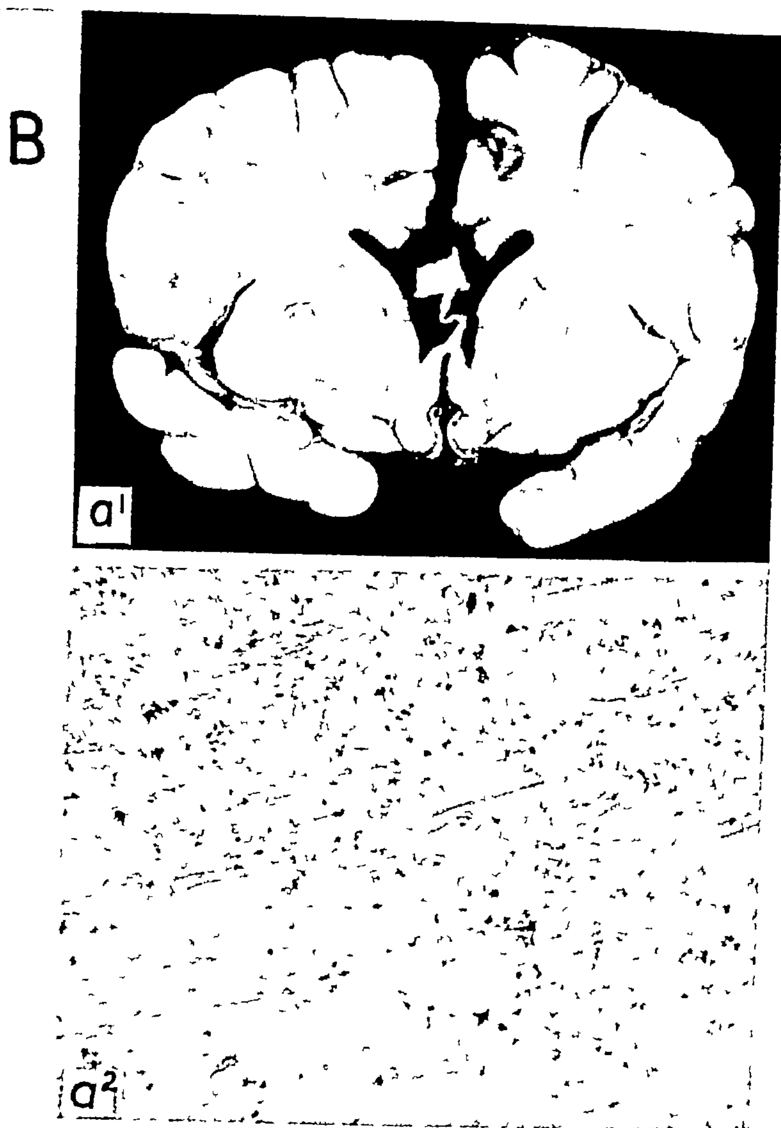


Fig. 68 (*continued*) (*B*) Grossly normal brain (a^1), fat emboli (a^2) revealed on microscopic section.

cases, the brain may appear fairly normal on cross section, but the presence of fat emboli and plugs of lipoid material can be demonstrated with special stains.

Diagnosis

The signs and symptoms of fat embolism are referable to involvement of the cardiorespiratory and cerebrovascular systems. Cough,

fever chest discomfort, dyspnea, fall in blood pressure, cyanosis, and pulmonary edema all point to the cardiorespiratory system. The cerebrovascular symptomatology ranges from headache and disorientation to coma and death and includes signs and symptoms suggesting intracranial hemorrhage, such as increasing loss of consciousness, convulsive seizures, and focal signs. Cutaneous petechiae may appear, usually on the chest, the extremities, and the conjunctivas. The number of petechiae has no bearing on the prognosis. Fat droplets may be found in the sputum, usually about 36 hours after onset of the embolism [19, 24] and in the urine 2 to 6 days after injury [20] However, fat has been found in the lungs of both fasting and lipemic animals[27] and in the sputum of patients with various conditions other than fat embolism [17] The urine to be examined should be obtained by catheterization and with complete emptying of the bladder, since the fat may be on the surface of the urinary fluid and remain in the bladder unless this is done.

Funduscopic examination occasionally reveals the presence of hemorrhages and exudates, in the form of patchy, whitish areas [22, 23] this is a particularly important sign if repeated examinations disclose the disappearance of old areas and the appearance of new ones.[12] Papilledema may be present.

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Chapter IX

POSTTRAUMATIC SEQUELAE

The sequelae of head injury may or may not be in direct proportion to the severity of the original trauma They range from death to a wide variety of neurologic and psychologic deficits The evolution of the various signs and symptoms is described in Chapter V In the usual deceleration injury, associated involvement of other organs and structures is often possible, so that the patterns of sequelae, based upon a combination of lesions, may be complicated

The pretraumatic personality of the individual is an extremely important factor in the posttraumatic picture A careful evaluation of any case must include an appraisal of the patient's pretraumatic state. Feelings of spite and resentment, and the possibility of compensation, also influence the posttraumatic picture

Death

Most deaths after acute head injury occur within the first 24 hours in both surgical and nonsurgical injuries despite intensive lifesaving efforts

When death occurs within a few minutes of head injury, the cause is undoubtedly involvement of the brain stem centers. Death of a patient comatose for several days after head injury is probably due to brain stem damage as a result of herniation of the hippocampal gyrus, or of herniation of the cerebellar tonsils at the foramen magnum Such herniations may be caused by cerebral edema or mass lesions. Cerebral edema, associated with swelling of the centrum ovale and the fiber tracts extending into the brain stem, is frequently caused by mass lesions compressing the brain. In many cases, respiratory paralysis occurs first, and is followed by vasomotor collapse.

The findings at autopsy of a series of 151 cases of fatal injury are shown in Table 12.

Akinetic Mutism[4]

Modern methods of treatment have made possible the maintenance of life in patients unconscious for prolonged periods. With proper care, some, particularly in the younger age groups recover without any residuals. In the older age groups, however, and especially in the presence of severe brain stem involvement the patient may continue in a vegetative state for months and eventually die. Some of these patients may appear conscious without any ability to communicate or respond intelligently to stimuli. Early, there may be decerebrate states, after several weeks, a more normal body tone and posture may return During the past few years, neuropathologic study of patients with this clinical picture has revealed the presence of devastating involvement of the greater portion of the cortex from causes other than trauma. The clinical picture of akinetic mutism may therefore be due to cortical involvement in certain cases. Strich[18] has reported extensive involvement of the subcortical white matter in certain cases of head injury with eventual death from intercurrent disease the clinical picture included decerebrate states with akinetic mutism

Table 12 *Autopsy Findings in 151 Cases of Fatal Head Injury*[54]

Data		*Number of cases*
Time of death		
On hospital admission		61
Within 10 hours		29
Within 24 hours		12
Skull fracture		
Single or multiple linear		83
Simple or open depressed		29
Extradural hematoma		
Surgical		11
Nonsurgical		4
Subdural hematoma		
Surgical		22
Right side	15	
Left side	7	
Ipsilateral skull fracture	7	
Contralateral skull fracture	8	
No skull fracture	7	
Nonsurgical		12
Right side	4	
Left side	3	
Bilateral	4	
Skull fracture	11	
Chronic		1
Subarachnoid hemorrhage		37
Intracerebral hematoma		
Massive		1
Petechial		71
Cerebral contusions		72

Spasticity, Paresis, and Paralysis (Fig 69)

The degree and type of permanent disability vary with the site and severity of the injury If the motor cortex is extensively damaged, the contralateral paralysis of the upper extremity may be permanent, whereas the initial paralysis of the lower extremity and possibly an associated speech defect may eventually improve. Speech disturbances resulting from lesions of the left side of the head may be severe to begin with, but recovery from a traumatic aphasia is fairly complete in many cases, unlike the aphasia of cerebrovascular disease and brain tumors.

Bilateral spasticities, decerebrate states, rigidities, and choreiform movements occur in lesions of the brain stem and particularly with lesions of the superior cerebellar peduncle red nuclei, and the cerebrospinal pathways. Bilateral cortical contusions and infarctions also may result in bilateral spasticities. In such cases, an inability to swallow and a nasal, pseudobulbar type of speech are common The former may improve rapidly, but the nasal speech may remain for many years. Parkinsonian like tremors may occur in the paralyzed or paretic extremity of the older patients.

The hemiplegias and hemipareses caused by extradural or subdural hematomas usually disappear more or less completely after the clot is removed In an occasional case, a static hemiplegia or hemiparesis may be associated with contractures difficult to overcome

Psychiatric and Mental Abnormalities

Disturbances of behavior the psyche, and mentation are common with head injury but in most cases they are not of a permanent character In evaluating the psychiatric effects of a head injury, the complete background of the patient — his age constitutional make-up, work and the site, type and severity of the injury — must be carefully correlated Psychiatric disturbances seem to be related to the

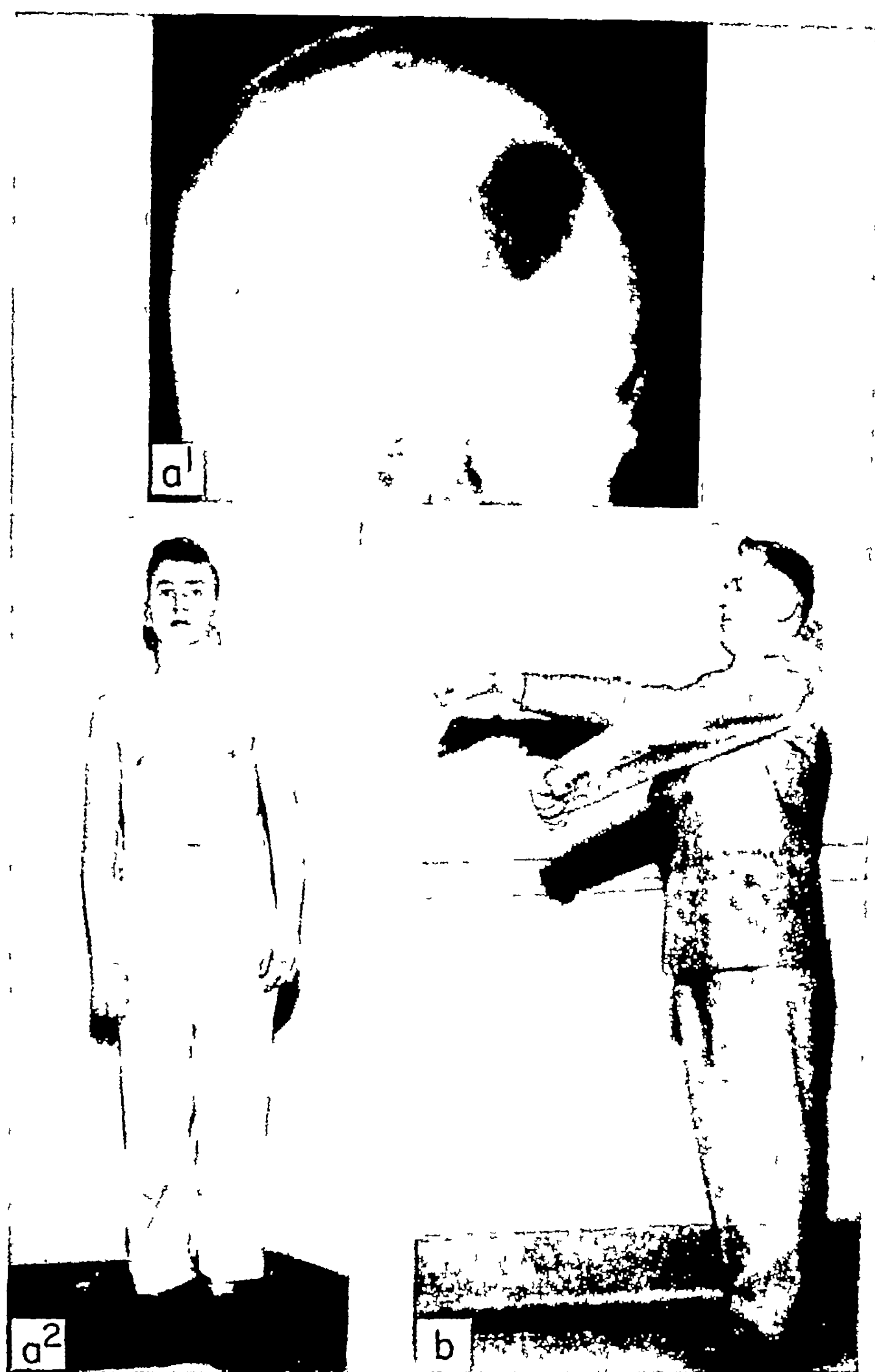


Fig 69 Hemiplegia in head injury (*a*) Right hemiplegia due to cerebral infarction after occlusion of left middle cerebral artery, upper limb most severely affected, left ventricle dilated (*a*[1]) (*b*) Left hemiplegia due to cortical laceration by shell fragments, upper limb more severely affected

diffuseness of the cerebral trauma, as manifested by the length of acute unconsciousness. Psychotic manifestations are less likely to occur with open head injuries than with closed ones associated with prolonged periods of unconsciousness In the latter case, even younger individuals may manifest profound and permanent deterioration. Posttraumatic psychiatric abnormalities may be classified as (*1*) severe psychiatric disturbances despite minimal injury (2) manic and confusional states following a period of unconsciousness, (*3*) retrograde amnesia, and amnesia for the period of severe mental disability (*4*) abnormal behavior due to severe cerebral injury with lowered mentation, (5) minor psychopathic states, such as traumatic neuroses, and (*6*) psychoses and psychoneuroses as a late sequel of head injury

A *severe psychiatric disturbance* soon after a minor head injury is probably due to the stress of the experience on a person with a pretraumatic neurotic potential When the abnormality appears some time after the injury, there is a good likelihood that the two are not related

The duration and severity of *manic and confusional states* following a period of unconsciousness depend on the nature and extent of injury Disorientation and confusion follow unconsciousness after trauma, and last 1 or 2 days to several weeks Judging by the fact that injury of the medial aspect of the temporal lobe in the course of exposing the circle of Willis causes a confusional and psychotic state lasting for several days, it seems probable that contusions of the limbic lobe, the subfrontal areas, and the tip and medial aspect of the temporal lobe (Fig 70) are a frequent cause of abnormal behavior in patients with head injuries.

With acute contusions of the temporal, frontal and limbic lobes, there may be disorientation and some degree of mania lasting several weeks. In such cases, the temperature is slightly elevated as a rule, and the cerebrospinal fluid is bloody Occasionally, there may be unilateral or bilateral circumoral twitching and unilateral or bilateral jacksonian seizures of the upper extremities.

a

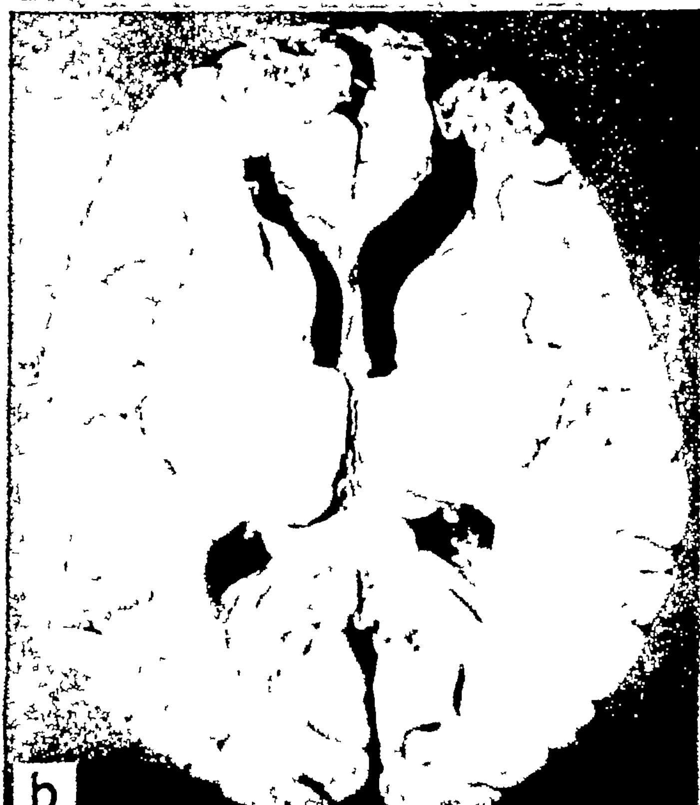




Fig. 70 Cerebral contusions resulting in scarring (*a*) Scarred orbital surfaces of frontal lobes, particularly right lobe, and some scarring of right temporal lobe, 20 years after acute head injury note "wormy" appearance of base of frontal lobe (*b*) Changes at tips of both frontal lobes, some cerebral discoloration, and hydrocephalus ex vacuo, more pronounced on right side frontal lobe injury of long standing (*c*) Contusions and scarring of left frontal lobe 3 areas of distinctly different ages of hemorrhage in 65 year old arteriosclerotic woman dead after the last of several falls oldest hemorrhage fills right lobe medially a more recent one somewhat laterally, and most recent one on superior and convex surfaces of hemisphere.

Occasionally, psychotic behavior after a head injury without loss of consciousness may be an indication of a hemispheral or posterior fossa hematoma. An outstanding and seemingly exaggerated symptom in these patients is the associated presence of severe headaches.

Retrograde amnesia shrinks as the patient recovers from the injury, and islands of memory may emerge in the course of recovery[19] The presence or absence of residual defects in recalling events before the injury may be used to evaluate recovery Memory defects may be due to intellectual loss as a result of the trauma.[23] Posttraumatic automatism ranging from 2 to 10 days after a period of unconsciousness is fairly common. The patient may respond and answer questions satisfactorily, but after recovery have a complete amnesia for this period

The *abnormal behavior* of some patients after severe head injury may be related to a lowered mentation. Psychiatric and psychologic tests have shown that the level of mentation, thought content, ability

to learn, and the amount of stored knowledge are definitely lower than normal in such cases It is therefore a justified conclusion that the anatomic cerebral connections are disrupted, resulting in less effective cerebral function The lower level of mentation may be a permanent sequel

Progressive mental deterioration after head injury is uncommon When it does occur, it is undoubtedly the result of severe injury of the central nervous system In such cases, the condition is apparently one of lack of cerebration rather than a psychoneurosis or psychosis

In the aged, severe head injury may result in serious, persistent mental deterioration, including confusion, disorientation, depression, unreliability, carelessness, or a total dementia Cerebrovascular disease (Fig 70*C*) would seem to be a predisposing cause, particularly in those with a history of stroke (*see* Fig 32)

Changes in personality traits after head injury have also been reported[8, 10] Personality disorders which develop some time after head injury may be due to chronic changes on the orbital surfaces of the frontal or temporal lobes. In general, however, psychiatric abnormalities after head injury tend to improve with time

Minor psychiatric abnormalities, such as the so-called traumatic neuroses, are common after head injury and can be explained on the basis of fright, pain, and anxiety about oneself, the family, or one's possessions In some cases, an inherent pretraumatic abnormality may be responsible for the nervous tension, headaches, and dizziness

The cause-and-effect relation of head injury to *delayed psychoses and neuroses* is difficult to evaluate precisely If the history reveals a normal intervening period, no cause-and-effect relation exists

Postconcussion and Postsubconcussion Syndromes

In our experience, both syndromes appear more commonly in women, and both are unusual under the age of 20 The main differ-

ence between the two syndromes is the absence of unconsciousness immediately after trauma in the latter and its presence in the former The clinical features are headache, dizziness, fatigue, nervousness, insomnia personality disorders, and poor memory Some symptoms are common complaints of patients in both groups, others are more or less unusual. Headache is a symptom in the vast majority, and in about 50 per cent there is both headache and dizziness. Smaller numbers complain of nervousness, fatigue, memory difficulties, or of pain in the neck and head Dizziness in the absence of headache, feelings of vertigo or rotation, and visual, hearing and olfactory disturbances are noted by small numbers of patients. An analysis of our series of 200 cases of these syndromes is given in Table 13

A chronic subdural hematoma or hygroma may be found occasionally Only rarely does the pneumoencephalogram suggest the presence of cerebral atrophy The electroencephalogram is normal in about 50 per cent of cases minor dysrhythmias may be found in the other 50 per cent. The skull roentgenograms are normal except for occasional skull fracture. Results of tests for labyrinthine dysfunction are usually normal, even when these reveal that the labyrinth is inactive, unilaterally or bilaterally, the patient may have only slight or no complaints referable to the labyrinthine mechanisms.

On the basis of observations of those with wide experience with head injury patients, it may be stated that (1) patients with major cerebral damage from closed or open injuries or with hematomas seldom manifest chronic headaches, dizziness, fatigue, or nervousness, (2) subconcussive or concussive blows, with a short period of unconsciousness or none whatever after impact are the most likely to produce either of the syndromes (3) the syndromes develop more commonly in tense anxious patients than in those with a stable personality (4) compensation considerations play a role in the duration of the symptoms.

The postsubconcussion syndrome is probably the result of functional and of organic cerebral disturbance due to a subconcussive injury Minor bruises and petechiae in important areas (subfrontal

TABLE 13 *Data on 100 Cases Each of Postconcussion and Postsubconcussion Syndromes*

Data	Number of cases			
	Postconcussion*		Postsubconcussion*	
Headache	81		91	
Unilateral		14		15
With dizziness		43		49
With vertigo		4		7
Neck pains	13		14	
Dizziness without headache	7		6	
Vertigo without headache	1		0	
Electroencephalographic features†				
Dysrhythmia grade I	19		24	
" II	3		2	
" I, with delta waves grade I	0		1	
Normal	35		37	
Labyrinth function tests‡				
Normal results	9		27	
In patients complaining of dizziness		7		16
Asymmetric or hyperactive results	5		5	
In patients complaining of dizziness		3		3

* Sex 66 male and 34 female, and 31 male and 69 female respectively

† Total of 57 and 64 patients, respectively, tested

‡ Total of 14 and 32 patients, respectively, tested

area, limbic lobe, hypothalamus) may cause confusion, a dazed feeling, and personality disorders despite the absence of any immediate posttraumatic unconsciousness The patient's concern and anxiety no doubt play an important role in the syndrome, but the psyche and its response to injury are the basic factors Concern and anxiety may be followed by minor symptoms, which in turn lead to more symptoms as the somatic functions affect the psyche

A condition described by Benon[1] in 1927 under the term "asthénie

chronique posttraumatique" resembles the syndromes under discussion here. He listed the symptoms as a feeling of weakness, fatigability, depression, headaches, pains in the limbs decreased desire to work, and increased need for sleep

It has been our experience that 25 per cent of patients with this syndrome recover completely within 1 month, and another 30 per cent within 6 months. In 20 per cent improvement occurs within 1 year, and in 10 per cent within 2 years. Complaints continue to be voiced after 2 years by 1 out of 10 patients, and a few have permanent residuals, such as anosmia or hearing defects

Posttraumatic Epilepsy (Figs 71-72)

Epilepsy as a sequel of craniocerebral trauma must be distinguished from the generalized or focal convulsive seizures which occur in the acute phase of severe head injury The location, severity and type of injury have considerable bearing both on the occurrence of epilepsy and on its incidence, the latter being much higher when the injury is at or near the motor cortex In the Russell and Whitty[17] series, parietal lobe injuries represented 65 per cent of the total number of cases with traumatic epilepsy

In closed head injuries, the incidence in several series has been reported as less than 5 per cent [12, 14, 15] in our experience, it is even lower — less than 4 per cent. Open head injuries have a greater incidence of epilepsy as a sequel between 15 and 22 per cent in civilian injuries (the higher figure applying to injuries with dural penetration), and from 18.9 to 45 per cent and over in war injuries. Not only dural penetration but intracranial inflammations and infections after head injury may be a factor in posttraumatic epilepsy In the Russell and Whitty series, the general incidence of epilepsy in a 5 year period was 45 per cent but in the cases with infection it was 54 per cent A summary of data on 100 of our patients with posttraumatic epilepsy is given in Table 14

A















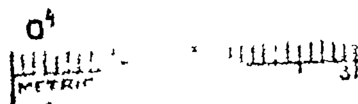


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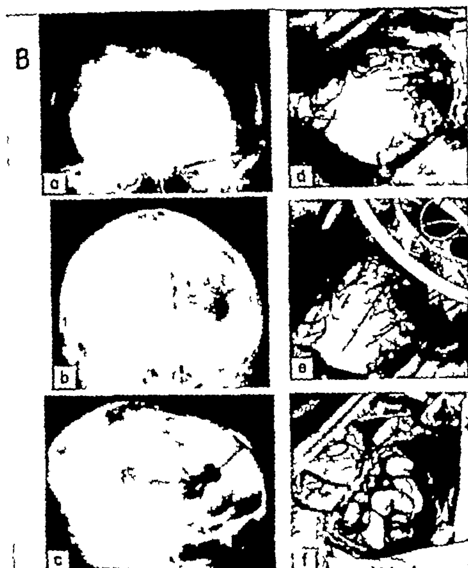


Fig 71 (*continued*) (*B*) Posttraumatic epilepsy due to injury in infancy (*a*) Asymmetric development of skull. (*b*) Dilated left ventricle and porencephalus. (*c*) Postoperative roentgenogram. (*d–e*) Tough piarachnoidal scar (*f*) Appearance after scar excision.

Pathophysiology and Pathology

There is general agreement that posttraumatic epilepsy has its seat in the cerebral cortex. Injury of the cerebral surface results in neuronal destruction and astrocytic and mesodermal proliferation The disappearance of oligodendroglial cells is directly proportional to the neu

A















Fig. 71 Posttraumatic epilepsy (*A*) First seizure 18 years after injury (a^1) Scar over site of open depressed fracture (a^2–a^3) Roentgenograms showing skull defect and bone fragments (a^4) Excised cortical tissue, consisting of scar and surrounding zone (a^5–a^6) Before and after excision of scar tissue (a^7) Electrocorticography (*see* Figure 72 for electrocorticogram).

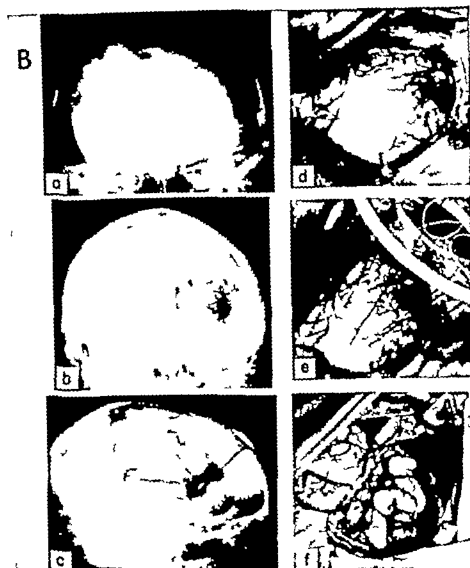


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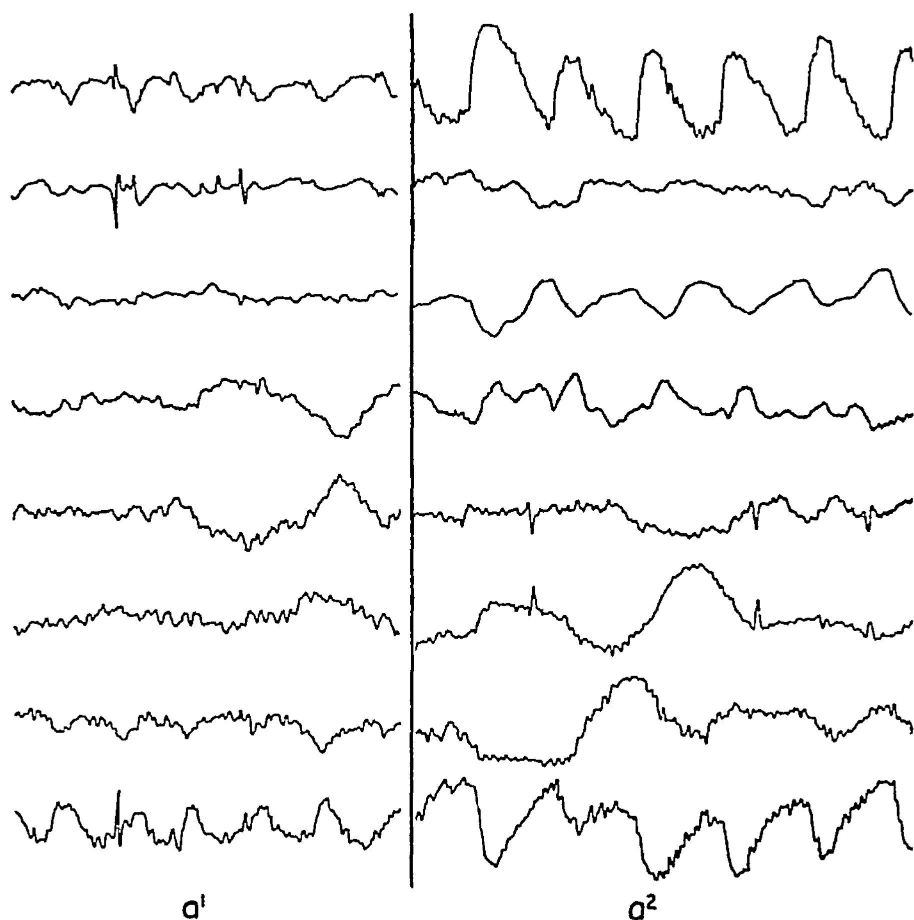


Fig 72 Electrocorticograms of case described in Figure 71 (*A*), spike discharges in leads 1, 2, and 8 along inferior aspect of scar before excision (a^1), these stopped after excision, but new spike discharges appeared on posterosuperior border of scar zone in leads 5 and 6 (a^2)

ronal destruction In open head injuries with destruction of cerebral tissue, an extensive cortical scar eventually develops and adhesions form between the dura and the scarred area The scar and its surrounding tissue then has a dual blood supply, the dural side being supplied by the external carotid artery, the pial side by the internal carotid This may be of some significance in the production of epilepsy, since neural vasomotor mechanisms are present in the distribution of the external carotid artery and are apparently absent or minimal in that of the internal carotid If the blood supply of the injured area is completely destroyed, cystic degeneration instead of cerebral scarring occurs

TABLE 14 *Data on 100 Cases of Posttraumatic Epilepsy*

	Number of cases		
Data	*Skull fracture**	*Concussion and subconcussion no fracture†*	*Mass lesions‡*
Unconsciousness on impact	26	27	3
Onset of convulsions			
0–3 months	12	16	2
4–6 "	8	6	1
7–12 "	9	10	3
13–24 "	10	5	2
3–5 years	4	2	—
6–10 "	5	1	1
11–18 "	3	—	—
Electroencephalogram			
Delta waves grade I	3	1	—
grade II	2	2	2
grade III	3	—	3
Dysrhythmia grade I	4	6	—
grade II	8	9	—
grade III	7	5	1
Normal	9	6	1

* Total, 51 cases 42 male, 9 female. Simple and open linear fractures, 12 simple depressed, 15 open depressed 24 Frontal 14 parietal 17 temporal 15 occipital 3 frontal sinus area, 2

† Total 40 cases 32 male, 8 female

‡ Total 9 cases 8 male, 1 female, Subdural hematoma 8 subdural abscess, 1

Contusions or hemorrhage deep within the brain may produce scars without meningeal involvement or adhesions between the dura and piarachnoid and in such cases the overlying piarachnoid may appear grossly normal *Eventually such lesions may* become cystic if they are sufficiently large to begin with

A meningocerebral scar is composed essentially of devitalized tissue, it is surrounded by an intermediate zone[12] of dead or dying cells (Fig

73), and finally by normal tissue. The size of the capillary network and the distribution of blood vessels in the three areas — scar, intermediate zone, and normal tissue — differ markedly The scar itself has no or practically no blood supply. The capillary network in the intermediate

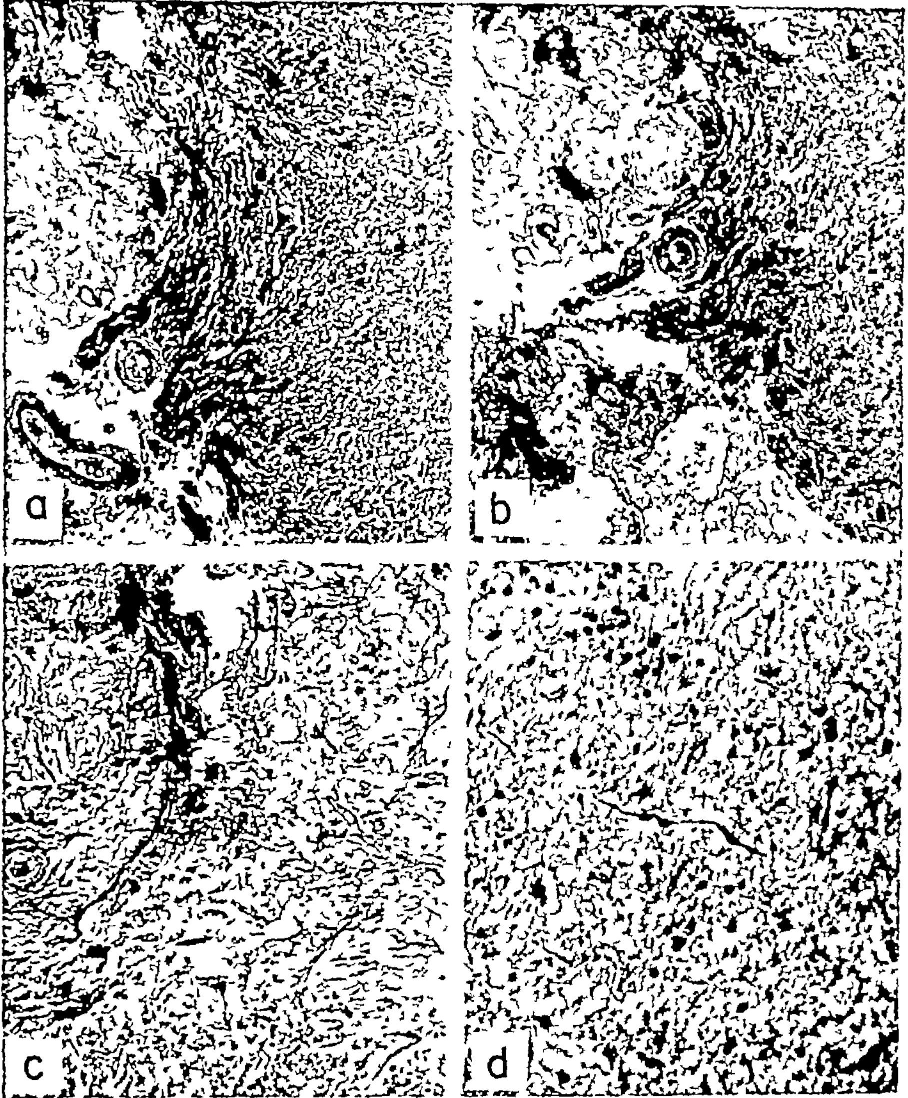


Fig. 73 Pathology of posttraumatic epilepsy, microphotographs of scar tissue (*a*) Vascularization of tissue, many blood vessels of external carotid artery distribution growing into brain tissue, hematoxylin–eosin stain (*b*) Preponderance of astrocytes (right) such gliosis may simulate microscopic picture of glioma or astrocytoma, glial stain (*c*) Mesothilial tissue, reticular stain (*d*) Swollen nerve fibers, Miller's fibrillar stain

zone is small, and is estimated to be a fiftieth of that in the normal tissue. The epileptogenic focus (or foci) is in the intermediate zone, usually in juxtaposition to the scar, but in some cases as far as 3 cm from the scar[20] Foci may be found not only on the affected side, but on the opposite side as well.

Penfield and Jasper[1] explain the late appearance of posttraumatic epilepsy in many cases as the result of further tissue destruction by the traction exerted by the scar on the intermediate zone. The gradual destruction of nerve cells in the zone, these authors suggest, may result in a low threshold for the initiation of a convulsive seizure. Another theory is that injury of suppressor areas destroys inhibitory impulses, thereby potentiating seizures[17]

Study of cortical metabolism during experimental epileptic seizures (metrazol induced) has shown that there is an increase in the cerebral content of lactic acid, in other words, an increased utilization of oxygen adenosine triphosphate remains at a constant level, phosphocreatine content decreases slightly[8] The potassium level is increased in the blood of the longitudinal sinus, and the sodium content of the neurons is proportionately increased during an epileptic seizure[2] Apparently the cerebral carbohydrate metabolism is adequately maintained during a seizure by an increase in blood flow and a somewhat higher oxygen tension

Extensive cavitation may be present in the vicinity of the cerebral scar, or a unilateral or bilateral ventricular enlargement. Evidence of cortical atrophy is found in some cases. Porencephalic cysts are found fairly frequently In our series of 100 cases of posttraumatic epilepsy, 39 were studied by pneumoencephalography the pathologic findings were generalized cerebral atrophy in 6, porencephalic cysts in 4, and unilateral ventricular enlargement in 2 in the remaining 27 the pneumoencephalograms were normal.

Microscopically, a scar usually consists of glial fibers (Fig 73) These epidermal derivatives may grow in juxtaposition to the mesodermal tissues, such as the dura piarachnoid, and the blood vessels, and exten

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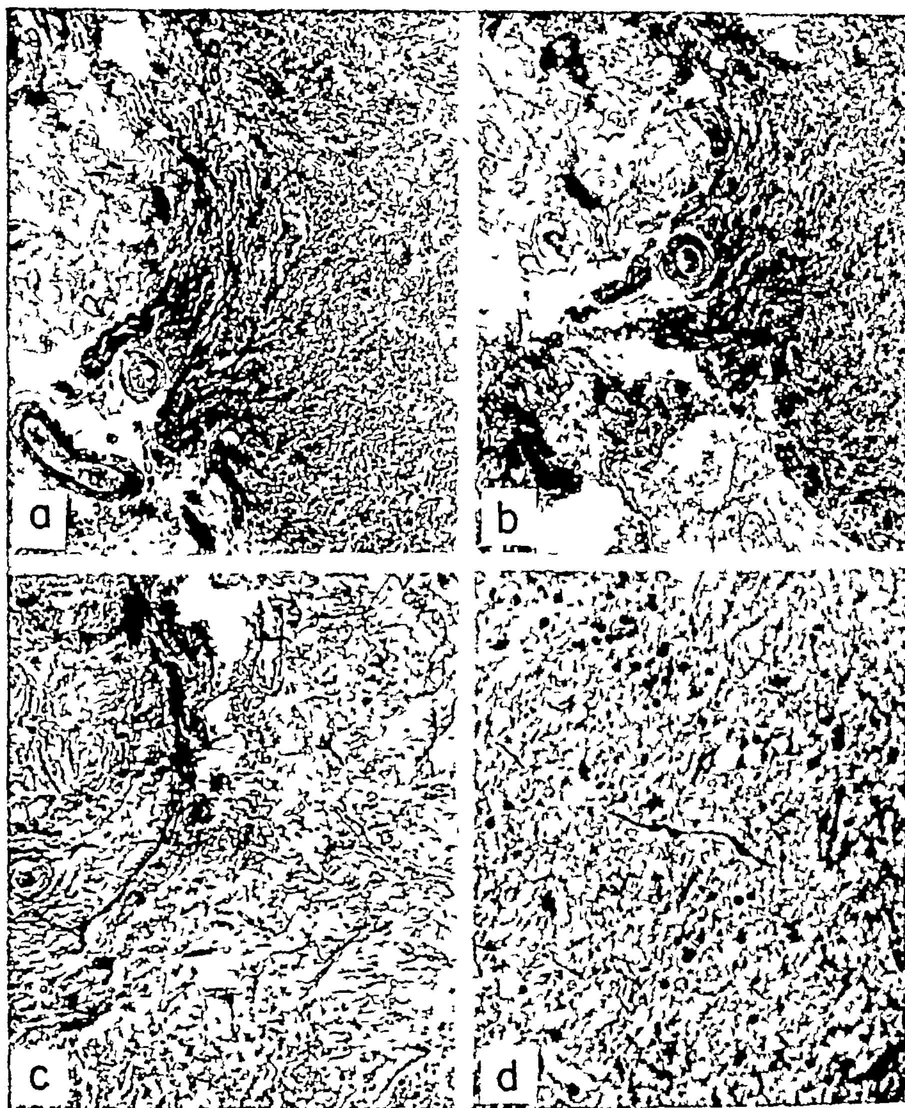


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sive adhesions may develop between membranes and the underlying brain Scarring is much more extensive when damaged cerebral tissue has not been removed, and extensive scarring is the rule in the presence of foreign bodies and organic matter, such as bone Large scars can also result from cerebral contusions not associated with open wounds (*see* Fig 70), and their microscopic features are essentially the same as those of scars resulting from open wounds.

Clinical Features

Usually, the first seizure occurs 3 to 18 months after injury Walker[20] is of the opinion that in 75 per cent of those who eventually become epileptic the first seizure occurs within 3 months of injury This has not been our experience In our series (Table 14), 30 had their initial attack within 3 months, 15 within 6 months or less, 22 within 12 months or less, 17 within 13 to 24 months of injury, and 16 after an interval of 3 to 18 years after trauma The interval between seizures ranges from 3 to 18 months in some cases, while in others a number of seizures may occur in rapid succession, possibly triggered by alcohol, constipation, psychic factors, and the like, after which many months or years may pass without a seizure Only occasionally do seizures occur during sleep

In many cases, the epilepsy is associated with focal signs and symptoms If the seizure is preceded by an aura, its motor or sensory manifestations may indicate the involved site. About 50 per cent of our cases have an aura, in most of these it is either sensory or motor, in some it is auditory, in a few visual and gastrointestinal In general, the seizures are jacksonian or generalized; an occasional patient has petit mal seizures Diencephalic and psychomotor seizures are rare The jacksonian and petit mal seizures occur more frequently in patients whose epilepsy is incompletely controlled by anticonvulsant therapy Frequently, these patients become sleepy after a seizure and, if undisturbed, may remain asleep for 1 to 6 or 8 hours Todd's paralysis occasionally appears after a seizure

Diagnosis

Electroencephalography may help to localize the epileptogenic focus or foci by the appearance of abnormal waves in the vicinity of a meningocerebral scar. Electroencephalography with metrazol activation (200 mg intravenously) may help uncover localized abnormalities in a little over half of the cases. Electrocorticography, which shows bursts of spiky waves in the epileptogenic zone adjacent to the scar but none from the scar itself may be of greater localizing value; such abnormalities were found in 14 of 39 cases of posttraumatic epilepsy.[21] However, since abnormal waves from the cortex may also be obtained at some distance from the scar,[11] an abnormal corticogram is perhaps not an absolutely reliable localizing aid. Cortical stimulation with a sine wave of 1.5 to 3 volts for 5 seconds during electrocorticography produces spiky waves for about 30 minutes. In some patients, this may be associated with the occurrence of an aura or a seizure. In one reported series,[20] about a third of the patients had no clinical symptoms of focal abnormalities during cortical stimulation, while the remainder had focal symptoms, and several among them a generalized seizure. Most of those without focal manifestations also did not have an aura.

On the whole, the electroencephalographic abnormalities are not pathognomonic of traumatic epilepsy. It has been suggested that the pattern of the EEG can foretell an oncoming attack.[1] In our experience, and apparently in that of Williams,[22] this has not proved to be the case.

Of the many abnormalities found in the pneumoencephalograms of patients with posttraumatic epilepsy, no one lesion is pathognomonic. Seizures may or may not occur in the presence of a porencephalic cyst; more common features are slight ventricular enlargement, or evidence of cortical atrophy, or extensive cerebral destruction, as evidenced by localized ventricular enlargement. It is noteworthy that the pneumoencephalograms of 27 of 39 patients so examined in our series were normal.

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tion to the body, and its prominence in comparison to the face The frontal and parietal eminences are large and conspicuous The infant skull is a nonrigid structure composed of loosely joined plates of pliable bone of uneven thickness attached to a rigid base and strengthened by the attachment of the dura mater and its associated septums The adjacent margins of the vault bones are separated by fontanels of fibrous tissue continuous with the dura internally and the pericranium externally The posterior fontanel is closed by the sixth week, the anterior by the ninth to the sixteenth month The sutures are not closed firmly until the fourth year

At first the internal aspect of the skull is smooth and quite firmly attached to the dura As the bone grows thicker, vascular channels and indentations appear Thus, the grooves for the middle meningeal artery and veins are absent in infants and are shallow in children

The frontal sinuses are practically absent at birth, while the ethmoid and maxillary sinuses are of fairly good size although they are not aerated The sphenoid sinus begins to form about the third year of life as an evagination of the posterior superior portion of the nasal cavity into the body of the sphenoid bone. The maxillary sinus is not fully developed until the age of 7

The important structural characteristics of the infant head, compared with that of the adult, are: (*1*) its elasticity and mobility, which depend on the degree of ossification of the cranial bones, (2) the shape of the skull and the fact that the bones are not of uniform thickness and strength, (*3*) the close attachment of dura to bone, the relationship of the various septums to the brain, the absence of the meningeal grooves or canals, and the presence of blood vessels in the dural septums

Through the age of 18 months, the chest circumference is about the same as that of the head, although somewhat smaller at first and somewhat larger by the age of 18 months Postnatal growth of the skull is somewhat slower than that of the rest of the skeleton[8] Most of the head's growth occurs in the first 2 to 3 years of life, then continues slowly to the age of 7, and accelerates again in adolescence The neural

portion of the head increases fivefold in volume, the facial structure about twelvefold between birth and maturity, and the cranial capacity from 400 cc. to 1,500 cc.

The circumference of the skull at various ages is [29]

Age months	*Male skull cm*	*Female skull cm*
Birth	35.3	34.7
6	44.0	42.9
12	47.1	45.9
18	48.8	47.4
24	49.6	48.2
30	50.1	49.0
36	50.4	49.3

Mechanisms of Head Injury

Information on the stress patterns of trauma in the infant head is lacking but important differences from those in the adult head may be assumed The infant skull being a pliable structure, can withstand a certain amount of deformation without injury of the intracranial contents. The elasticity of the infantile cranial vault makes it possible for much of the energy of an injuring object to be absorbed by local deformation so that little is transferred to the intracranial contents. The characteristics of both static and dynamic loading in the infant skull are probably not the same as in the adult skull, and greater energy would be required for serious injury The absence of buttresses and the uneven ossification of the vault serve to explain why skull fractures in the infant are often irregular infrequently linear, and without predictable pattern. A localized injury frequently results in a "derby hat" type of depression The fragmentation of bone so characteristic of depressed fractures in the adult is largely prevented in the infant by the membranous character of the parietal areas in the infantile skull. With increasing age the stress characteristics begin to approach those of the adult, and after the age of 2 to 3 years linear fractures occur, the di

rection apparently depending on the presence of the buttresses (Fig. 74) Posttraumatic unconsciousness is probably less common in the infant than in the adult because the energy of the blow is largely dissipated in deforming the skull, little being transmitted intracranially to involve the brain stem centers On the other hand, extensive cranial deformation may tear the dura from the brain, with resultant hemorrhage and cerebral contusion.

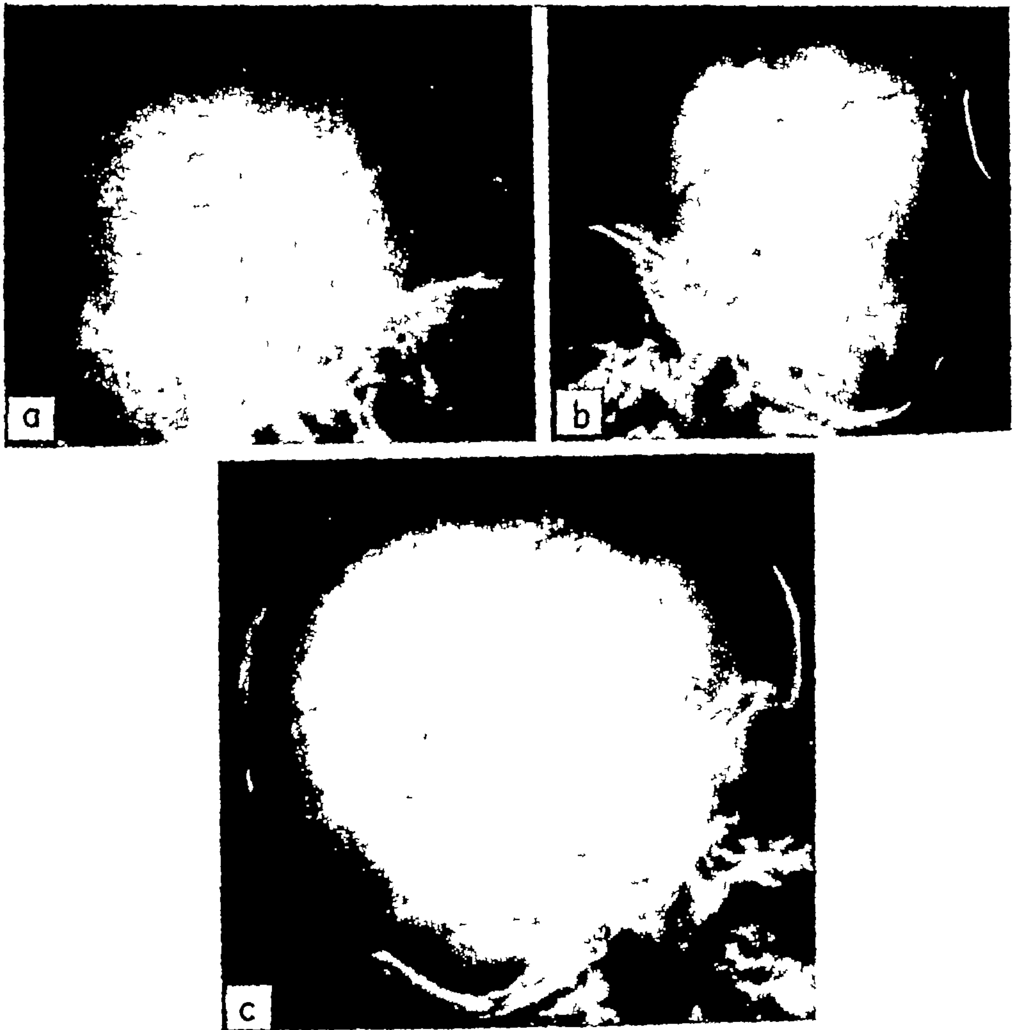


Fig 74 Skull fractures in children and infants (*a*) Parietotemporal fracture in 4½ year old child, note adult pattern (*b*) Temporal fracture in 8 year old child; impact area outlined by wire, note adult pattern (*c*) Linear parietal bone fracture in 6 month old infant, cerebral compression by extradural hematoma.

Injury at birth may occur as a result of the birth process itself, or of other factors The uterine contractions, which force the fetal head against the resisting birth canal, can compress the easily yielding fetal skull when the head is engaged in the pelvis, the compression may act to shorten the long diameter of the head The forces of the birth process can also produce shearing and stretching

Distortion of the bony structure of the head may stretch the dural septums, such as the falx cerebri and the tentorium.[23] If the distortion is severe, the tentorium may be torn at its junction with the falx Veins in this area, including the great vein of Galen, may be disrupted, with a resulting hemorrhage. The free edge of the tentorium easily compresses the brain stem with possible contusions, lacerations, and hemorrhages. In a series of 167 fetal deaths during labor, the tentorium was torn in 81 and in 5 of these the falx as well subdural hemorrhages, varying from a few drops to copious extravasations, were present in all but 6 cases the tributaries to the great vein of Galen seemed to be the important source of the hemorrhages.[15] Others believe that a more common source of the bleeding is the veins to the sagittal sinus, disruption of which causes extensive unilateral or bilateral subdural hemorrhages.[18]

Instruments used in delivery are another source of cranial injury in the newborn. Improper application of forceps may cause intracranial hemorrhages and in some cases simple depressed fractures (derby hat type) (Fig 75) Of 9 cases of such fractures in our experience, only 1 had a dural tear and intracranial damage was slight or nonexistent.

While injury of the pregnant woman at full term often poses a medicolegal problem we have found that such injury hastens labor but in most cases does not harm the infant.

Birth Injury

Cranial trauma in the newborn is most frequently the result of injury during the birth process. The trauma varies from minor and

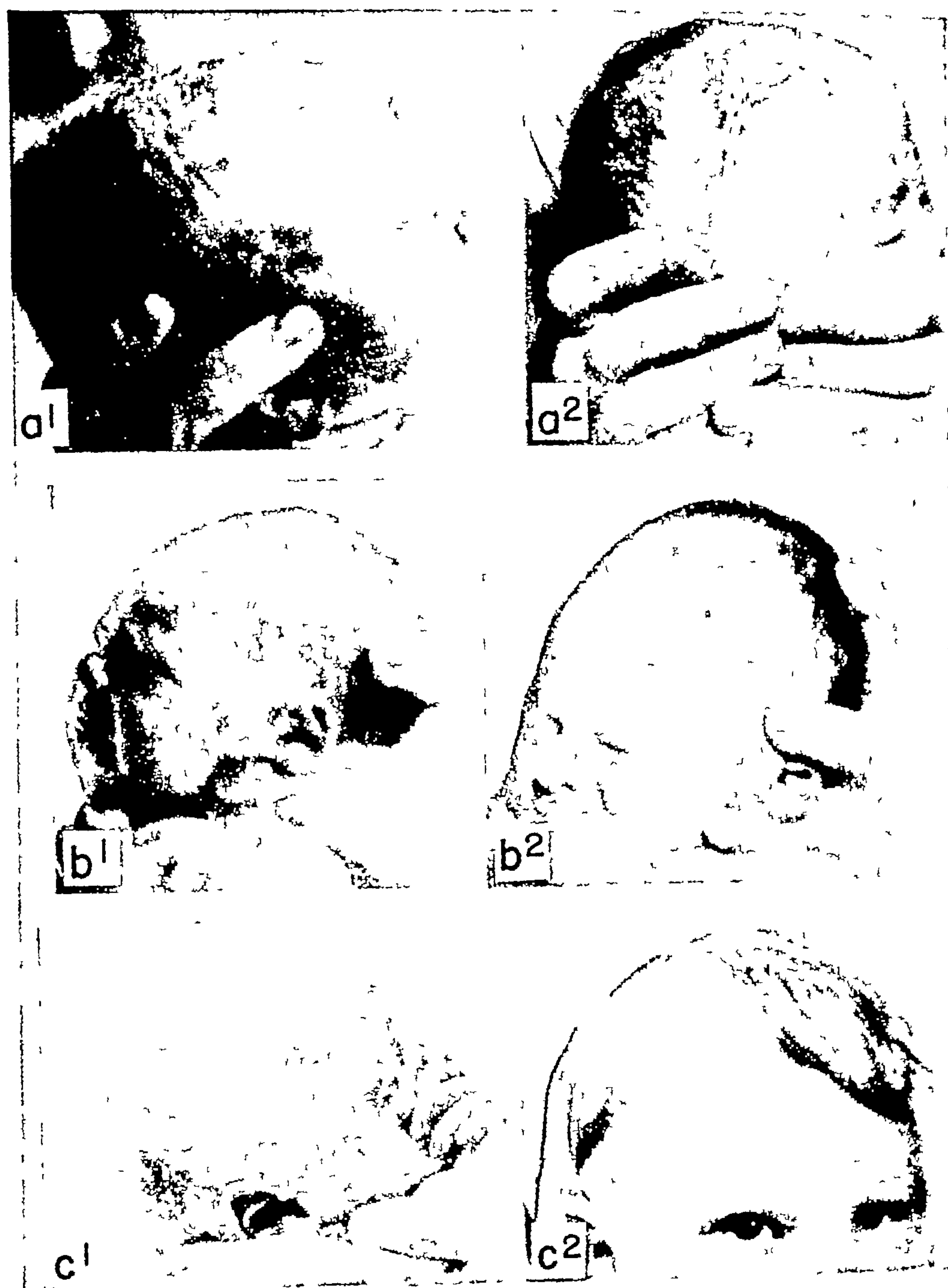


Fig 75. Depressed skull fractures in newborn infants after forceps delivery (*a*) Area of depressed bone, and incision through which depressed bone was elevated (*b*) Midline frontal depressed fracture, elevated through curved incision. (*c*) Depressed fracture at right parietofrontal junction, and child 2 years later.

clinically insignificant to severe involvement with permanent neurologic sequelae.

The importance of traumatic intracranial hemorrhage as a cause of cerebral damage in the newborn has been emphasized repeatedly In infants dying at birth or within the first 2 weeks, hemorrhages are found in a large percentage, but only in 33 per cent of them is the hemorrhage severe enough to be the cause of the death[1] As reported in the literature, the incidence of intracranial hemorrhage in the still born ranges between 13 and 69 per cent[12] Most commonly the hemorrhage is over the hemispheral convexities in about half the cases, the hemorrhage is unilateral, in the remaining half bilateral with that on one side much larger than on the other Hemorrhages at the base of the skull in the posterior fossa and about the upper end of the cervical cord are found fairly often, particularly in the more seriously injured infants.

Blood in the cerebrospinal fluid was found in 60 of 473 newborn infants in one study of consecutive births, only 26 of the 60 had clinical signs of cerebral involvement, and 12 of them died postnatal life in the remaining 34 was normal and without obvious clinical abnormalities.[20]

Extradural hematoma in the newborn is a rare occurrence, but a few such cases have been reported[2, 3, 18] Almost always, the hematoma is the result of a skull fracture and a torn blood vessel Campbell and Cohen[3] describe an example of an extradural hematoma due to a depressed fracture caused by forceps the infant had a large clot over the cerebellum as well as over the occipital portion of the hemisphere on one side. Subdural hematoma usually bilateral, on the other hand is fairly common[1, 14, 20, 22, 23] In many infants with skull distortion the hemorrhages are small and some of these hemorrhages may be asymptomatic. But when the distortion causes tears of dural sinuses or the great vein of Galen the subdural hematoma may be large. The disruption of the last named structure may cause extensive extravasations of blood over the brain stem and in the posterior fossa.

The major congenital neurologic deficits are those of the motor

system (spasticities and paralyses) and of mentation (varying degrees of intellectual impairment) The relative importance of birth injury, asphyxia, anoxia, prematurity, and developmental factors in the etiology of these deficits is difficult to establish According to Alpers,[1] the role of anoxia has been overemphasized, and it would seem that birth injury, in itself, is not the most important cause. For example, of 200 infants with bilateral spastic paralysis, only 15 per cent had been delivered with abnormal labor, 33 per cent were premature, and in 35 per cent the head was smaller than normal [12] In a series of 100 cases of mental deficiency that came to autopsy, birth injury accounted for 30 to 35 per cent of the idiot group, but only 8 per cent of the imbecile group, and a still lower percentage of the high-grade moron group [3]

The pathologic abnormalities which may be found in infants surviving intracranial injury at birth include (*1*) cortical lesions eventually leading to local or general atrophy or to cyst formation; (2) ventricular distortions; (*3*) subdural hematoma, at times, a cystic mass enclosed in a fibrous covering between the dura and the arachnoid; (*4*) calcified small hematomas, in later years identifiable on skull roentgenograms,[11] (5) thickening of the arachnoid, with obliteration of the subarachnoid spaces and in some cases an external hydrocephalus, (*6*) porencephalic cysts, sometimes communicating with the ventricle or subarachnoid space, as a result of an intracerebral hematoma or infarction.

Motor and intellectual deficits in the newborn, as well as epilepsy, may also be caused by symmetric atrophy of the hemispheres,[6] by lobar agenesis, with sclerosis and microgyria,[4] asphyxia,[27] and other lesions which are difficult to identify because of the presence of mixed pathologic states

Penfield and Jasper[24] believe that the effects of childbirth may have a bearing on the later appearance of epilepsy They suggest that cerebral compression with resultant ischemia, may injure the medial aspect of the temporal lobe near the uncus and the hippocampus by *unilateral* or bilateral herniation against the incisural border(s). They also suggest that cerebral ischemia may lead to the development of microgyria dur-

ing pregnancy In our opinion, agenetic mechanisms and congenital causes are more important in the causation of microgyria than localized damage due to vascular insufficiency Local areas of damage usually result in cystic degeneration, whereas a microgyric area usually has the cortical structural characteristics, although sclerosed and smaller than normally We also feel that microgyric areas are probably developed early, about the third or fourth month of gestation.

Skull Fractures

Simple linear fractures may occur in infants and in children, and the older the child the greater the likelihood of adult fracture patterns (Fig. 74) An occipital bone fracture due to a fall upon the occiput is usually linear, extends to the foramen magnum, or lateral to it, and appears in the midline or on either side of the foramen magnum. In this type of fracture, cerebral involvement is usually mild or absent.

Diastatic fractures are more common in infants than in adults, and the extensive and widened fracture line may simulate the bursting type of adult fracture. Many of the diastatic fractures undoubtedly result from the incomplete calcification of the vault bones and their lack of uniform tensile strength. Usually, the patient s clinical state is less severe than might be expected from the extent of the skull deformation resulting in the fracture Apparently severe skull deformation followed by fracture can occur without obvious signs of involvement of the intracranial contents in the young child or infant. Often, when the patient is first examined there is little evidence of serious injury, only roentgenography reveals the fracture indicating that there must have been a severe impact. Diastatic separations of suture lines, such as the lambdoid suture are important in the formation of extradural hematoma.[3] Venous hemorrhage from an emissary vein or the lateral sinus may cause formation of a large hematoma

Simple depressed fractures are fairly common in infants and children because their bones are still membranous. In most, there is only slight

or no associated damage to the intracranial contents. Dural laceration may occur with diastatic as well as with comminuted fractures. The edges of the fractured bone may also shear the dura from the skull When the arachnoid is also torn, cerebrospinal fluid escapes through the fracture into the subaponeurotic areas, collects beneath the scalp, and slowly increases in size When aspiration yields cerebrospinal fluid instead of the more usual thick, black, stagnant blood of a hematoma, a spurious meningocele (Fig 76) due to an arachnoidal tear is present. In general, repeated aspiration is of little use, and the wiser procedure is exposure of the dural and arachnoidal lacerations and closure by water-tight suture.



Fig. 76 Spurious meningocele after comminuted fracture, removal of bone fragments and closure of dural defect with temporal fascia led to good recovery.

On palpation, a small hematoma beneath the scalp may simulate a depression in the skull In such cases, roentgenography is important to establish the correct diagnosis

In the infant, the skull may grow normally after a derby-hat type of depressed fracture has been elevated (Fig 77), or the injured bone may be completely absorbed, with complete lack of calcification in the fracture area. No definite cause for the bone absorption has been found, but in most cases the explanation that arachnoidal cysts erode the margin of the fracture [illegible] ble one.

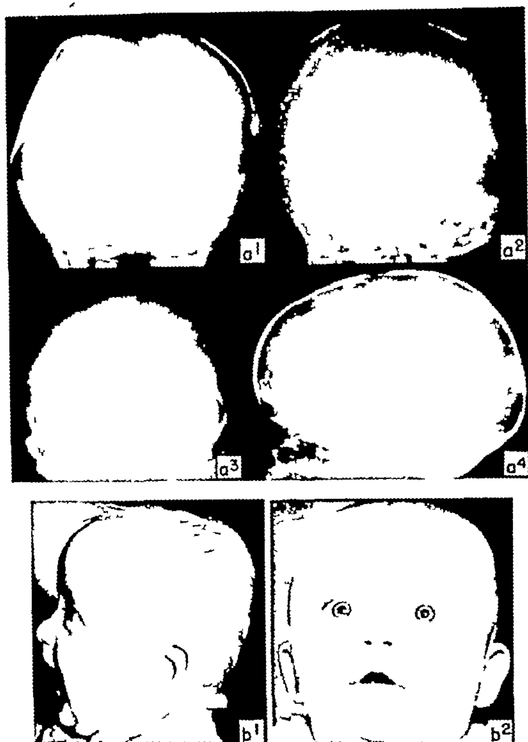


Fig 77 Depressed skull fractures in newborn infants after forceps delivery (*a*) Before after and 4 years after elevation of depressed bone. (*b*) Infant (*see also* Figure 79*a*) with flame hemorrhages in both retinas, slightly unequal pupils, and bilateral subdural hematomas.

Penetrating injuries, in view of the child's propensity to play with pointed objects (sticks, pencils, umbrella stays, knives, sharp-edged toys), occur more often in the child than in the adult, war excepted An apparently minor scalp wound may therefore be associated with injury to the skull and its contents A detailed, accurate history, roentgenography, and exploration of the wound may be necessary to determine the extent of involvement We have seen cases of penetrating injuries with cerebral involvement in which the first symptoms were caused by increased intracranial pressure due to a brain abscess (Fig. 78); only by a postoperative reconstruction of the events was the his-

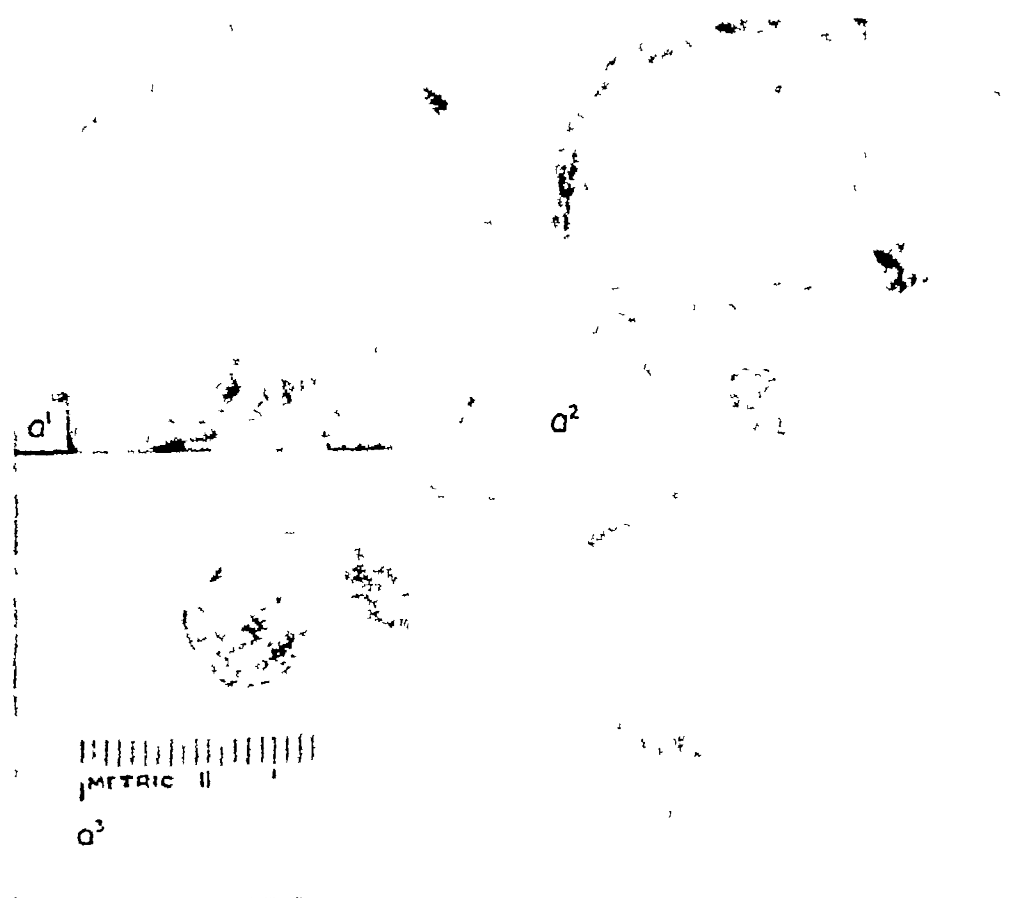


Fig. 78. Open, dep… frontal a… puration over fracture a… …ve roentg… and stone (a^1) which had… …ull for al…

tory of the injury determined The principles of treating open head injuries in the child are the same as for the adult (*see* Chapter XIV)

Hematomas

Cephalhematoma

This is a common finding in infants it usually occurs in the parietal area, although we have also seen it in the frontal and occipital regions. It is almost always unilateral, but occasionally may be bilateral It is a subperiosteal hemorrhage, so that its lateral extensions are controlled by the attachment of the periosteum to the connective tissue between the lamellas of bone and the mass is therefore localized to a single lamella. It may be absorbed rather rapidly, and disappear completely in the course of 2 to 3 months. In some cases, portions of the mass may harden or even calcify We have seen 1 case in which an extensive osteomyelitis of the skull developed after attempts to evacuate the clot with a syringe and a needle. A cephalhematoma should not be mistaken for a subaponeurotic hemorrhage of the scalp

Extradural Hematoma

Extradural hematoma in the newborn has already been discussed It is rare in infants, and unusual in children. Of 13 cases in our experience under the age of 12, only 1 was in an infant below the age of 6 months. In a reported series of 30 cases under the age of 12, 15 were in children less than 2 years old[8]

The small, clinically unimportant extradural hematomas which often occur with depressed skull fractures and are usually due to bleeding from diploic and smaller dural vessels are not considered true extradural hematomas in the sense in which the term is used in this text.

Usually the hematoma is the result of a low velocity deceleration impact such as occurs in falls to the sidewalk in bicycle accidents, or in falls from a short distance The following are examples of extradural hematoma in young children

Penetrating injuries, in view of the child's propensity to play with pointed objects (sticks, pencils, umbrella stays, knives, sharp-edged toys), occur more often in the child than in the adult, war excepted An apparently minor scalp wound may therefore be associated with injury to the skull and its contents A detailed, accurate history, roentgenography, and exploration of the wound may be necessary to determine the extent of involvement We have seen cases of penetrating injuries with cerebral involvement in which the first symptoms were caused by increased intracranial pressure due to a brain abscess (Fig. 78); only by a postoperative reconstruction of the events was the his-

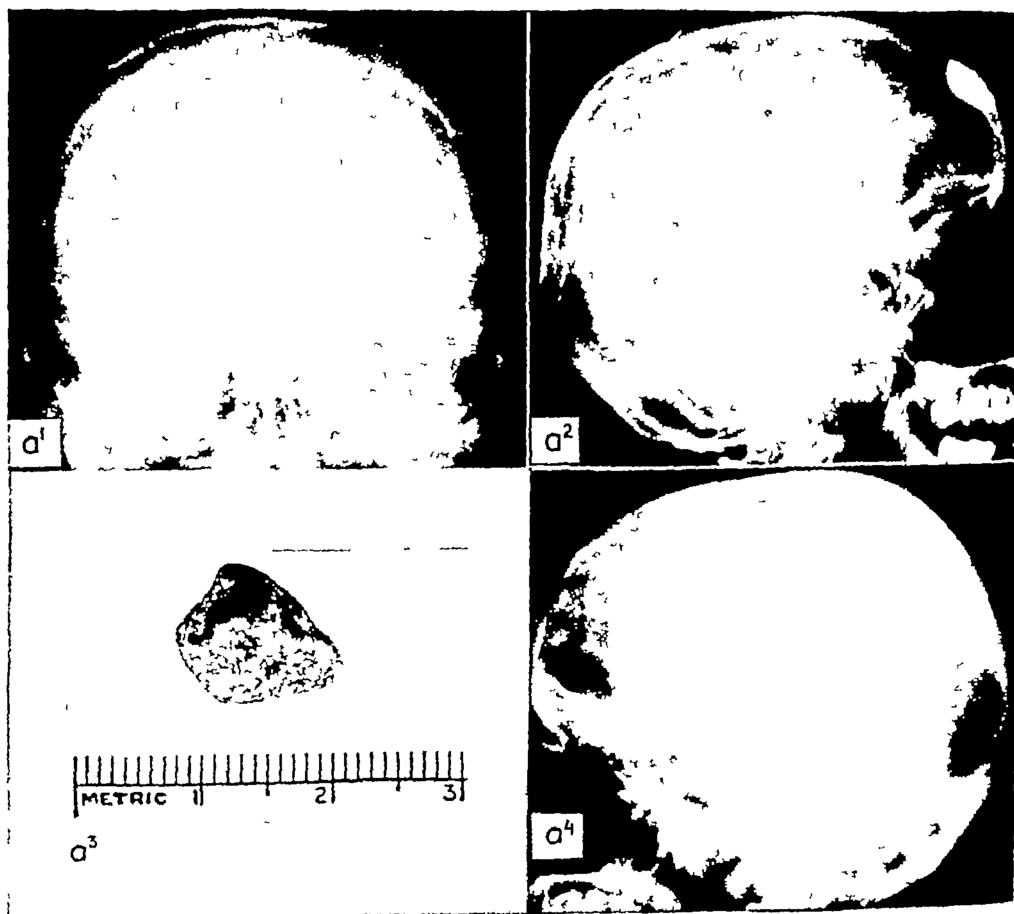


Fig. 78. Open, depressed skull fracture in left frontal area in adolescent, suppuration over fracture area Pre- and postoperative roentgenograms (a^1–a^2, a^4), and stone (a^3) which had remained embedded in skull for about 10 weeks.

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A 14 month old child fell a distance of 18 feet, 10 hours later the child became unconscious, a left parietotemporal hematoma was removed and the child recovered[30] A 13 month old child fell a distance of 4 feet, 2 hours later the child became unconscious and twitching of the left side of the body—face, arm, and leg—appeared, somewhat later, a left hemiplegia became apparent, with ocular deviation to the right, a large parietotemporal hematoma was removed[14] A 3½ year old child died 12 hours after falling off a swing, at autopsy, a right middle meningeal hemorrhage and a skull fracture were found[17] In our series, a 3½ month old child fell out of the crib, 10 hours later increasing stupor with weakness of the right half of the body developed, the roentgenogram showed a diastatic fracture of the left parietal bone, an extradural hematoma under the fracture site was removed and the child recovered

There may be an initial period of unconsciousness, or the child may be able to walk away from the scene of the accident, although in a somewhat dazed condition Within several hours, headache of increasing severity develops, the child begins to vomit, becomes somewhat somnolent and then stuporous, and eventually there is respiratory arrest if the clot is not evacuated With progressing cerebral compression, cortical, cranial nerve, and brain stem manifestations usually appear Convulsions are an early sign A dilated pupil, usually on the ipsilateral side, may develop, with a contralateral hemiparesis or hemiplegia In theory, the cerebrospinal fluid should be clear, but in practice it is often bloody, the pressure may not be high, although in some cases it is definitely above normal In the infant, the usual signs of compression may be masked by a shocklike state, with weak and rapid pulse, pallor, and a low temperature as a result of blood loss into the extradural area[16] Surgical evacuation must be attempted in such cases to prevent the development of an irreversible state terminating in death

Extradural hematoma in children with diffuse cerebral damage may present a diagnostic problem On admission to the hospital, the injured child may not have a lucid interval, and signs of deterioration and cerebral compression may be present In the absence of a temporal

bone fracture, a mass lesion can only be suspected, and its type and location only presumed The hematoma may be extradural, subdural, or intracerebral Exploratory trephination may be necessary to establish the diagnosis, if time permits, angiography in older children may be of diagnostic help

The skull roentgenograms may not show a fracture, or there may be a diastatic fracture, particularly of the lambdoid suture. Venous bleeding from emissary veins may result in a large extradural clot in the infant,[8] which is not the case in adults

Subdural Hematoma

A subdural collection of blood, the hematoma or of cerebrospinal fluid the hygroma is more common in infants than in children The young infant may tolerate a subdural collection better than an older child or an adult because of the elasticity of his skull The infant may appear hydrocephalic, but his general demeanor is nearer normal, he responds better to his environment, and the head does not enlarge to the extremes usual with hydrocephalus. Percussion of the skull may give a "cracked pot resonance. A tight anterior fontanel is common. Focal signs may be present. At times the infant may be malnourished and there is a history of poor feeding and vomiting

The causes of subdural hematoma in young infants are birth injury or injury in falls and vehicular accidents. Chronic subdural hematoma has also been reported after surgical treatment of hydrocephalus.[9]

A juvenile, relapsing type of chronic subdural hematoma (Fig 79) has been described in children who have recovered from a subdural hematoma in infancy or early childhood.[11] In such children, bony overgrowths, consisting of an elevation of the sphenoid ridge, superior orbital plate, and the middle fossa, and a hypertrophy of the frontal and ethmoid sinuses may be seen These overgrowths result from the natural absorption of the clot, and a lower than normal pressure in the area. If a second injury occurs, hemorrhage into the area of the first clot will produce severe signs and symptoms in such cases, the possi

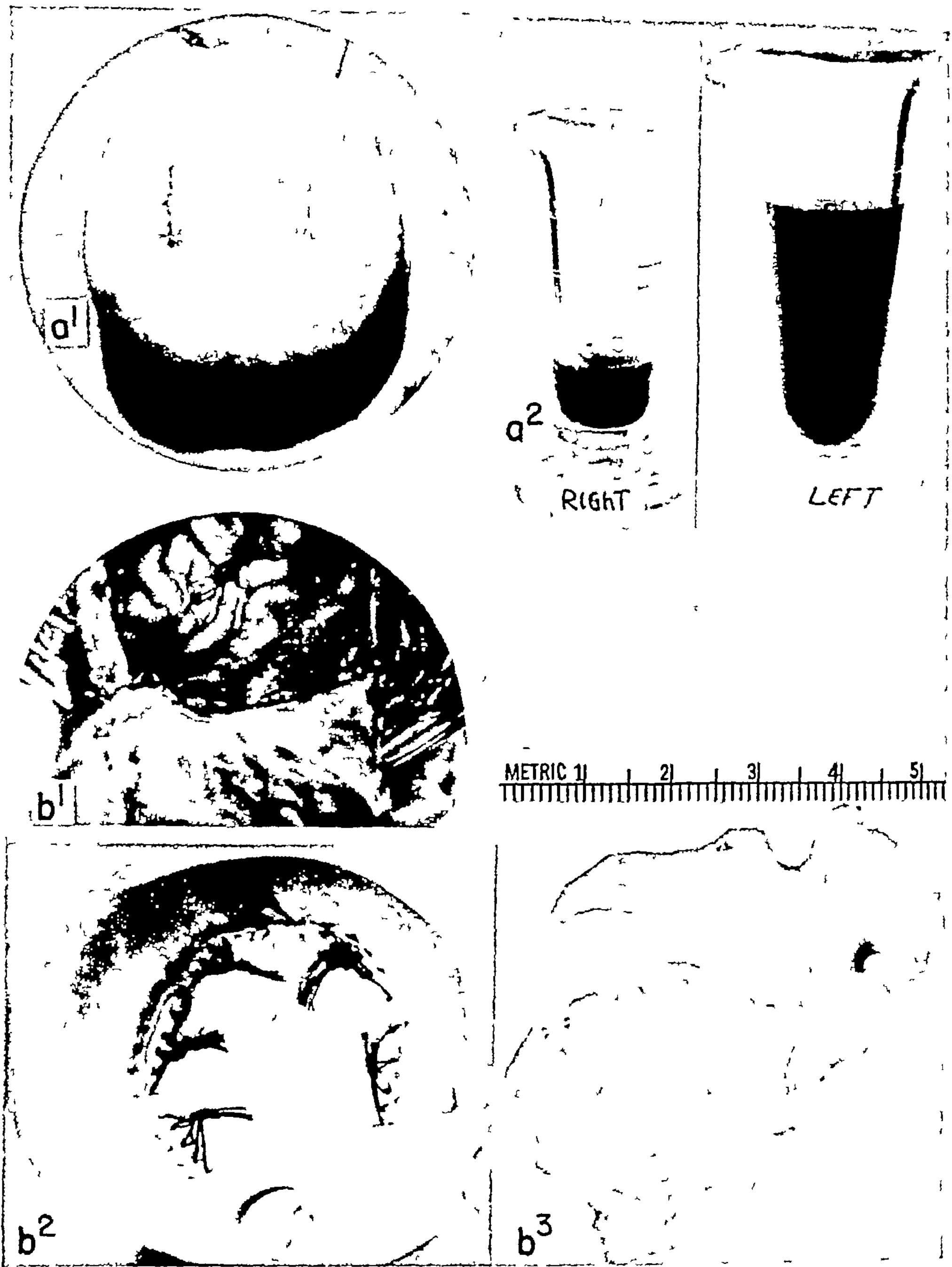


Fig 79 Subdural hematomas in infants (*a*) Burr openings and bilateral liquid hematomas removed through them (*see also* Figure 77*b*) (*b*) Subdural hematoma with membrane, (b^1) *in situ*, (b^2) closure after osteoplastic craniotomy, (b^3) membrane

bility of a second hematoma should be considered when the skull roentgenograms reveal unequal development. It is noteworthy that the below normal pressure of cerebral agenesis does not cause hypertrophy, in many such cases the skull being smaller on the side of the agenesis than on the other side.

Particularly in infants and young children, severe head injury with development of subdural hematoma may affect the further development of the cranial and intracranial structures. In the patient illustrated in Figure 80 an injury at the age of 11 weeks had resulted in bilateral subdural hematomas. After the hematomas were drained, pneumoencephalograms demonstrated considerable cerebral atrophy The head developed asymmetrically, and an internal hydrocephalus and mental retardation were present Convolutional markings in the more posterior portions of the head suggested the presence of localized pressure in this area

The pathologic features of subdural hematoma in the infant are somewhat different from those in the adult. In a majority the hematomas are bilateral and are over the frontoparietotemporal region In some, they may also extend into the occipital area, thus covering the entire hemisphere. The consistency and color of the blood and its residuals are also not the same as in the adult, in the acute case, the hematoma is fluid and may be bright red, later the hematoma becomes brownish or chocolate colored A mixture of clotted and liquid blood may be found or the clot may be organized, or even calcified The chronic hematoma may be completely liquid, and enclosed in a membrane which is thick on the dural side, thin on the arachnoidal side. In some, the contents may be xanthochromic, or almost clear, but with a high total protein content.

The hygroma (hydroma) is a collection of clear or xanthochromic cerebrospinal fluid It may or may not be a residual of injury Collections of cerebrospinal fluid whether xanthochromic, clear but with increased protein content or normal may form in the subdural space as a result of abnormal cerebral development This condition which



Fig. 80. Bilateral subdural hematomas in infant, removed through trephine opening, (a^1–a^2) 1 year later, poor physical and mental development, markedly enlarged ventricles and microcephaly of left side of skull; (a^3–a^4) pneumoencephalograms 6 months apart; postoperative roentgenogram (a^5) and 1 year later (a^6), showing definite change in shape of skull.

has been described as external hydrocephalus, may be mistakenly diagnosed as the result of injury, mainly because the fluid contains blood or its breakdown products.

Subdural tap through a 20 gage needle in the lateral angle of the anterior fontanel may yield bloody or xanthochromic fluid Repeated taps may be indicated in the very ill infant. Later, burr openings may be made for better drainage of the contents, if there is a membrane, an osteoplastic craniotomy is necessary, in order to remove as much of the membrane as possible as well as the fluid contents.

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Chapter XI

HEAD INJURIES IN SPORTS

The nature of the sport usually determines the type of injury incurred Sports can be divided into two large categories· those requiring body contact, and those employing objects which serve as missiles or as striking objects Obviously, a few sports, for example, baseball or hockey, fall into both categories The injuries in some sports, such as automobile racing or hunting, differ so little from those caused by automobile accidents or guns in the general population as to require no special consideration The likelihood of head injury is much greater in certain sports than in others, these include boxing, baseball, golf, and football, with boxing having the greatest incidence

Boxing

A study of the mortality in boxing showed that in a 6 year period (1946-1952) there were 88 deaths, 58 of professional boxers and 37 of amateur boxers,[18] the mortality rate, on the basis of an estimated 2,000,000 bouts, is 0 004 per cent In New York State, there were 7 deaths between the years 1946 and 1953, in New York City, 21 deaths in a 32

year period [7] in Detroit, 1 death in approximately 400 bouts between 1949 and 1954.

The *mechanisms* of head injury in boxing are fairly complex. The energy of blows delivered to the head may vary from 5.6 to 17.4 foot pounds or more of kinetic energy light heavyweights, using 8 ounce gloves, average 17.4 foot pounds per punch, flyweights (112 pounds or less), 5 6 foot pounds, heavyweights, using 6 ounce championship gloves, 26.2 foot pounds, and lightweights, 8 7 foot pounds.[8]

From 750 to 1,700 blows may be administered in a fight going to 10 rounds, most of them obviously, not delivered to the head Complete or partial loss of consciousness may result not only from direct blows to the head but also from blows injuring the neck structures, the carotid sinus, the chest, the heart, or the solar plexus. Among the causes of unconsciousness with direct head blows are

(*1*) Injury of one or both labyrinths by a blow to the head or lower jaw involving the petrous bone, the resulting loss of equilibrium resembles an unconscious or semiconscious state. This may be a frequent cause of the knock-out in boxing according to Sherrington [11]

(2) Injury of the posterior hypothalamus or upper brain stem, milder injuries produce some disturbance of the conscious state, with sluggishness, grogginess, amnesia and weariness. The more serious injuries cause unconsciousness lasting for hours or days before improvement occurs, or the unconsciousness continues until death The milder blows may be considered as subconcussive, the more serious ones as concussive. Cerebral contusions and petechial hemorrhages in this area are possible without skull fracture or other cerebral injury [2]

(*3*) Injury to the brain as a result of a sudden rise in intracranial pressure due to the blow, mass movements of the intracranial contents, and the effects of repeated blows to the head, the injury may be mild and reversible, or serious and irreversible, possibly ending in death. There is experimental evidence that repeated minor blows to the head can cause extensive injury of the brain and hemorrhages.[10, 12, 13]

Blows to the neck in the region of the carotid sinus may cause

syncope, accompanied by pallor, weakness, and bradycardia. Dilatation of the blood vessels, with a precipitous fall in blood pressure, may occur in some, while in others there is a temporary cardiac arrest. The unconsciousness resulting from a carotid sinus reflex resembles that of head injury.

The solar plexus blow causes unconsciousness by deranging the vagal tone of the body. A paralysis of the vagal inhibitory activity causes a rapid heart beat and a rise in blood pressure, stimulation of vagal activity, on the other hand, slows the heart rate, reduces the cardiac output, and decreases the blood pressure. Intense stimulation may possibly cause heart block, or cardiac arrest. This type of injury may also cause breathlessness or apnea. The unconsciousness from a blow to the solar plexus or the carotid sinus may result from vasomotor dysfuntion, with a reduced oxygen supply to the brain, or from impulses extending into the brain stem centers through vagal and glossopharyngeal connections. In most cases, however, the unconsciousness is probably of vascular origin.

Experimental studies on frogs,[6] rabbits,[20] and dogs[20] provide the basis for the explanation of the effects of solar plexus blows or chest blows in the region of the heart. In the frog, repeated abdominal blows resulted in stoppage of the heart in diastole, when the vagi were cut, this did not occur. In the rabbit and dog, repeated blows to the lower left chest wall caused a serious drop in blood pressure, and the electrocardiographic pattern became disorganized.

An "exhaustion syndrome,"[16] with a rise in the pulse rate, drowsiness, and finally circulatory collapse and coma, is a distinct possibility in boxing. For at least 6 hours before a fight, many boxers limit their food and fluid intake, others may become seriously dehydrated in their attempt to lower their weight to the proper figure for the final weigh-in. The violent exertions which then follow, under the heat of the ring lights, and often at high temperatures and high humidity, may cause a serious loss of sodium chloride from the blood, resulting in a dis-

turbance of the conscious state. In others, the rise in body temperature during a fight cannot be controlled in the normal manner by sweating because of the dehydration in such cases, dysfunction of the heat regulating center may occur In one championship match this syndrome was not recognized by the experienced seconds and trainers.[18]

The *asthénie chronique posttraumatique* which Boigey[1] mentions in his book on sport injuries may possibly be related to the exhaustion syndrome.

The *signs and symptoms* of head injury in boxing depend upon the seriousness of the injury, and the resulting pathology They include (*1*) varying degrees of unconsciousness (*2*) the manifestations of intracranial hemorrhage — extradural or subdural hematoma, subarachnoid hemorrhage, or petechial brain stem hemorrhages (*see* Chapter V), (*3*) conjunctival intraocular or retinal hemorrhages, paralyses of extraocular muscles, difference in size of pupils (*4*) signs of increased intracranial pressure.

Roentgenographic evidence of skull fracture is unusual except in cases in which the boxer has fallen to the floor in extension A fracture of the occiput or other area of the skull may occur as a result of secondary deceleration Blows to the jaw may fracture the temporal bone in the area of the condyloid fossa particularly if the temporal bone is thin (use of a mouthpiece now greatly minimizes the chance of such injury)

Electroencephalographic abnormalities in boxers have been reported by some investigators,[2] and denied by others.[11] In our experience, there may be no correlation between the severity of the injury and the electroencephalographic pattern soon after injury, but in serious injury definite abnormalities appear several hours or days later

Subdural hemorrhage or hematoma is the commonest cause of death in boxing In 21 deaths in New York City over a 32 year period, subdural hematomas were the cause of death in 15 death occurred within 40 hours in 10, and in 2 to 4 days in 5.[7] Our only fatal case was also due

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to a subdural collection with an extensive pontine hemorrhage; death occurred 18 hours after injury. Another cause of death is petechial hemorrhages in the brain stem or elsewhere in the cerebral tissue

The term "punch drunk," first used by Martland,[17] is commonly applied to a late posttraumatic syndrome in boxers The condition is characterized by mental deterioration, slurred speech, waddling gait, and a parkinsonian tremor The cause has been ascribed to repeated cerebral injuries, and to petechial hemorrhages[5, 10] Definite pathologic data and a careful clinical analysis of the syndrome are lacking.[12] In the 2 cases of the syndrome in our experience, the pneumoencephalograms revealed some evidence of cerebral or cerebellar atrophy. In the one with cerebellar atrophy, an unsteady gait developed 22 years after a bout with Joe Louis, since he later showed improvement, it is possible that his unsteadiness had a hysterical basis The second patient, who had been in many fights and had been knocked out repeatedly, talked "as if he had pebbles in his mouth," had generalized tremors, walked with a slightly unsteady and propulsive gait, and had a staring expression The pneumoencephalograms showed some generalized cerebral atrophy, and slight ventricular enlargement with some increase in the size of the subarachnoid spaces In a report on 2 other cases of the punch drunk syndrome, the pneumoencephalogram showed no abnormality in one, and cerebral atrophy over the left frontal area in the other[4]

Anosmia, or loss of the sense of smell, which according to Boigey[1] is a common complaint of boxers, is not a feature of boxing injuries in our experience

In evaluating mental abnormalities in a boxer, the possibility that they antedated the boxing injury instead of being caused by it must be borne in mind His mental age, educational status, family history, and psychiatric disturbances must be considered in the diagnosis of the punch drunk syndrome, as well as other injuries or other organic diseases which might cause the abnormalities However, since mass movement of intracranial contents, with resultant cerebral contusions,

and hemorrhages do occur in boxing and since such lesions may cause mental abnormalities, it is possible that boxing can cause mental defects.

Other Sports

Severe head injury is more frequent in baseball than in any other sport except boxing The series of head injuries in sports in a 32 year period, collected by Gonzales,[7] gives 43 deaths from baseball injuries, 6 from football, 3 from handball and squash, 2 from skiing and 1 each from basketball and golf. Johansen,[11] in a recent report, lists 31 cases of head injury in skiing in a 2 year period In our experience, there have been 4 cases of severe head injury in golf, 1 of them being fatal

Head injury in baseball ranges from minor to severe (Fig 81) A direct blow by ball or bat, collision between players, or collision with an obstacle, are all sources of head injury A well pitched ball, which weighs about 6 ounces, may travel at a speed of 100 feet per second and have an impact energy of some 650 inch pounds.

In golf (Fig 81), injury is inflicted by a direct blow of golf ball or club The ball may strike any part of the head, but the club injuries are usually in the frontal area and as a rule are incurred by caddies.

In hockey, a direct blow by a hockey stick, or falls and collisions, are the usual causes of head injury, but most of the injuries are not severe.[9] In football, there may be a direct blow by a kick, or blows in falls and collisions. The latter are also the main causes of injury in handball and squash

In most of the sports, acceleration and deceleration plays a role, depending on the primary cause and the violence of the sport.

Various types of skull fracture may occur in baseball and golf, including linear fractures and open or closed depressed fractures, which may be of the "pond type. As with any head injury, there may be cerebral laceration or contusion and extradural or subdural hematoma The subdural hematoma may occur in the absence of skull fracture,

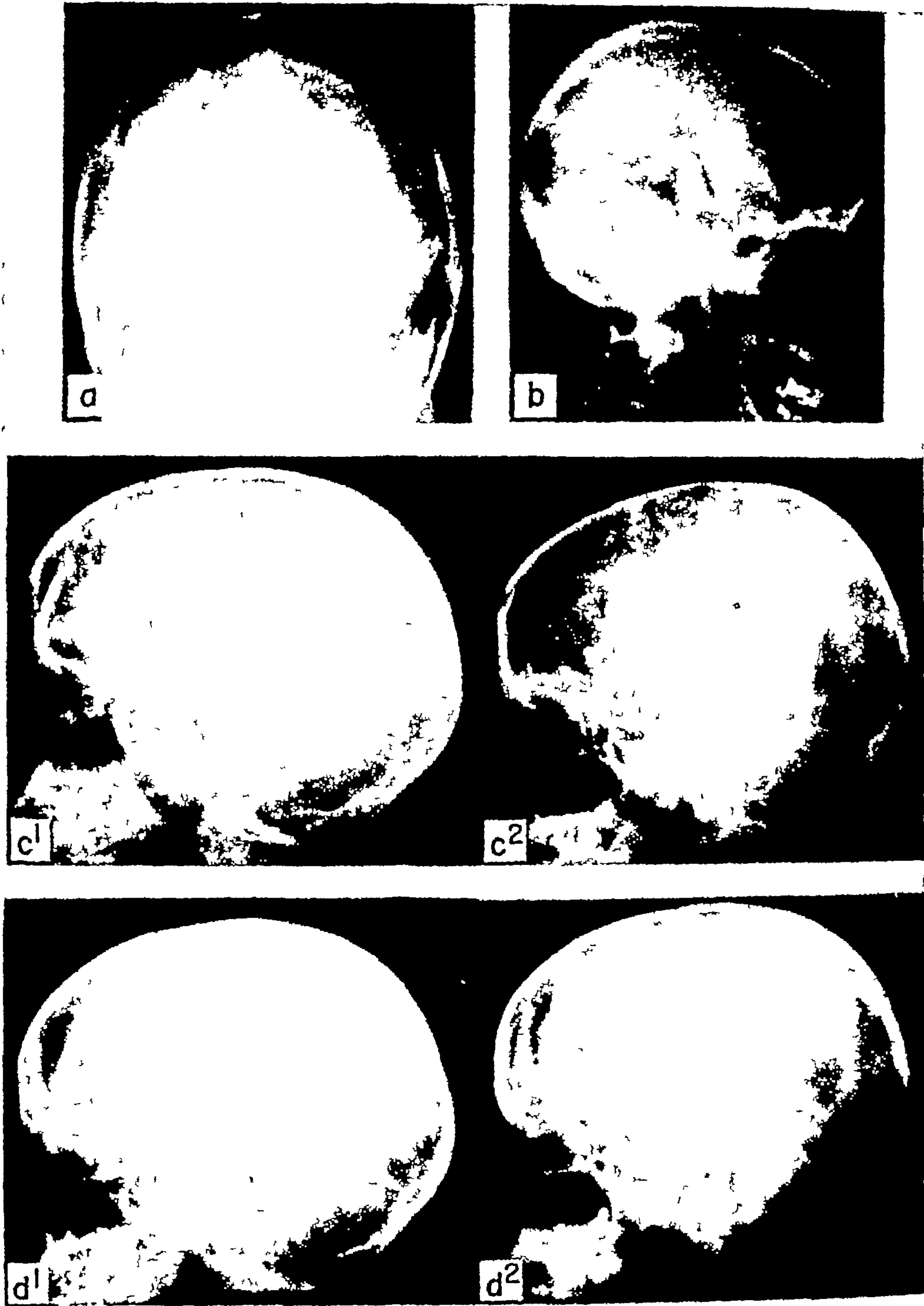


Fig 81 Head injuries in sports (*a–b*) Baseball injuries: depressed fracture with fragmentation of bone (*c–d*) Golf injuries, open depressed fractures of frontal bone by golf club, in both cases, bone fragment was elevated and patients recovered without deformity in forehead

and skull fracture is not necessarily accompanied by unconsciousness. Concussion rather than fracture may occur in golfing accidents In the minor baseball injuries, posttraumatic states may last for several weeks.

Many of the football injuries, in our experience, are of the mild concussive or subconcussive types the head is not a focus of purposeful injury, as it is in boxing and is reasonably well protected by the helmet. In severe injury, there may be skull fracture, and extradural or subdural hematoma with or without fracture. Most of the injuries sustained in hockey are lacerations and contusions of the face and scalp

The pathologic changes the diagnostic methods, the symptoms and signs, and the treatment of head injury in sports, aside from boxing are no different from those of any other head injuries, and are described *in detail in other chapters.*

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Chapter XII

MEDICOLEGAL ASPECTS OF HEAD INJURY

Physicians are often called upon for court testimony as ordinary or expert witnesses, particularly in cases involving workmen's compensation. So far as the physician is concerned, the legal aspects of head injury are limited to a careful and comprehensive evaluation of the patient's complaints and disabilities, an accurate diagnosis, a determination of the existing degree of disability, or a prognosis of it, and as precise and fair a presentation of the information as possible.

The evaluation of a head injury in a person involved in litigation is based on the physician's accurate observations and well-documented statements. In giving his testimony, he should have the confidence arising from a thorough study of the patient, who is now the plaintiff. As a witness, the physician needs only a rudimentary knowledge of court procedure. Particularly in compensation cases, the lawyers of both sides are interested primarily in the degree and permanence of the disability and the relation between the events leading to the injury and the person's present state. The physician's statements about the patient's physical, mental, and psychologic disabilities at the time of the medical examination may become part of the court record.

The method of eliciting information by examination, both by the defendant's and the plaintiff's lawyers, often serves to confuse the medical witness In giving his testimony, the physician can best fulfill his responsibility by adhering strictly to the facts as he knows them· by presenting them precisely, avoiding, at the same time, medical terms and complicated explanations which would be confusing; and by maintaining the demeanor of an impartial specialist

Evaluation of Disability

When a person submits to a medical examination for an injury which is in litigation, he waives the privilege of the confidential relation between patient and doctor which obtains ordinarily Although ethically the results of the examination might be considered the exclusive property of the one who has employed the physician to carry out the examination, in practice the information also becomes available to the opposing attorney via legal channels

The progress of the litigation and the justice of the settlement may depend to a considerable degree upon the skill and thoroughness of the medical examination, and the conclusions reached by the examiner. The history of the accident that has caused the injury should be recorded in detail, and should include, if possible, a description of the injuring object, its speed or force, the height from which the object or the patient fell, and so on, all such data help to estimate the potential of the injuring force Time must be taken to assess properly the disability suffered immediately after the injury and later, questions about the time lost from work, the attempts to work, and the requirements of the job all provide pertinent data The evaluation of the patient's mental and psychologic characteristics and his pretraumatic background, revealed only as the patient tells the complete story, sometimes haltingly, requires time, but is important. Such a painstaking history may not be admitted as testimony later, and objections may be

raised that it is hearsay evidence or even grossly untrue It is valuable to the medical examiner, however, since it helps him to form an opinion about the injury and the resulting disabilities

The physical and neurologic examinations must be as thorough as possible and performed with the patient unclothed The scalp and skull are scrutinized carefully, and any abnormalities noted The status of the cranial nerve functions, the fundi, the visual fields, motor power, sensation, reflexes, coordination, speech, gait, mental and psychologic reactions must all be tested, and the results recorded The various diagnostic technics (pneumoencephalography, ventriculography, angiography electroencephalography, myelography) may have to be used to arrive at a correct evaluation of the clinical diagnosis. Evidence of recent or old disease may be found, unreported in the history, which may have a bearing on posttraumatic complaints, as, for example, a complaint of disabling headache after a minor head injury by a litigant who has hypertension The educational background should be noted in every case, there are a surprising number (native-born or alien) who are still illiterate or nearly so, lacking any formal education The examiner, since he is responsible for the clinical diagnosis must determine whether the claimant is simulating or exaggerating a disability whether he is malingering, hysterical, neurotic, or psychotic. For example, in examining for loss of hearing acuity, it should be borne in mind that test responses are subjective, in the absence of definite knowledge of the patient s preaccident hearing, however, the loss must be presumed to be due to the accident.

It may be necessary to call upon various specialists — in ophthalmology, psychiatry, otology internal medicine, endocrinology, roentgenography, orthopedics — to confirm or clarify the diagnosis. In general recommendations for such examinations should be included in the examiner s report Some attorneys prefer a report which contains only the findings referable to the central nervous system, and that opinions about mental derangement be made by a psychiatrist. In any

event, the report of the examination must describe, clearly and completely, all objective clinical neurologic findings which are related to the diagnosis and the disability

Disability

Medically, a disability is a physical or mental derangement of normal function Legally, disability is defined as an inability to perform normal productive activity, or, "a state of being disabled from earning full wages at the work in which the employee was last subjected to the conditions resulting in disability" "Pain and suffering" are important issues in cases other than workmen's compensation, and may be compensable on the basis of severity and duration, in compensation cases, this issue apparently does not influence decisions, especially when the claimant has been able to continue his work

Disability is classified as (*1*) temporary total, (2) temporary partial, (*3*) partial permanent, and (*4*) total permanent This is an accepted classification, and is used as a basis for determining medical care and settlements Certain injuries are held to result in total or partial disability, and a schedule of compensation is part of most Workmen's Compensation laws Michigan, for example, lists the following as total and permanent disability. (*1*) total and permanent loss of the sight of both eyes, (2) loss of both legs or both feet at or above the ankle; (*3*) loss of both arms or both hands at or above the wrist; (*4*) loss of any two of the members or faculties enumerated above; (5) permanent and complete paralysis of both legs, or both arms, or of a leg and arm, (*6*) incurable insanity or imbecility A disability not falling into one of these categories is considered a temporary total disability for a period of not more than 500 weeks from the date of inj... Permanent and total disability, as defined, is conclusively pre... to continue for 800 weeks from the date of the disability or the ... loss; if the capacity to earn wages has not been re-established ... end of 800 weeks, the claimant can present his case to the Wo...

Compensation Commission and secure further benefits for an indefinite period up to the end of his life.

Most Workmen's Compensation laws specify that reasonable medical surgical, and hospital services and medicines be provided when they are needed for a specified time, with additional periods upon application to the Commission. They also specify that the employer must supply appliances which may be necessary to cure or relieve the effects of the injury, for example, hearing aids artificial limbs, glasses, artificial eyes, teeth. The laws also provide for the reporting of an accident, how long after an accident a claim for disability can be made, and various other details.

A person claiming disability may be requested by the employer or the insurance company to undergo a medical examination from time to time, and has the right to provide and pay for the presence of his own physician at the examination

The physician examining a claimant should be familiar with some of the provisions of the Workmen's Compensation Law of his state regarding disability and compensation. A common example of the type of case an examiner encounters is that of a worker who, after a cranial injury, insists that he is suffering from headache, dizziness, fatigue, and nervousness, and refuses to work on a factory assembly line, the physician must establish the validity of these symptoms, evaluate their basis, and determine the prognosis He may also be called upon to treat the patient and submit progress reports which may determine the continuance of compensation benefits

The examiner may also have to determine the relation between preexisting occupational disease and disability or death as well as disability or death resulting from an accident to a worker with a disease that is not occupational, such as a brain tumor A worker who has a brain tumor may slip in the course of his work, and in falling may strike his head a resulting hemorrhage into the tumor may cause prolonged disability or even death. If the clinical and pathologic factors in this case are correctly and completely reconstructed, and

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fairly presented, a just settlement is possible under the Workmen's Compensation Law of many states

The provision in most compensation laws that a complication in the course of a compensable injury or any added disability or death becomes compensable includes any error or act of malpractice by the physician treating the employee

In summary, the problem of determining the degree of disability after head injury is more complex than after most other types of accident The compensations for some disabilities, for example, the loss of a limb, a finger, or a toe, are specified in the law But a person who has suffered a head injury may have incapacitating symptoms for a long time, despite the absence of objective neurologic deficits Head injuries may cause psychiatric derangements which may be an aggravation of a pre-existing neurosis On the other hand, total and permanent disability resulting from the accident may be unquestionable in a severe head injury In open head injuries, the possibility of post-traumatic epilepsy, with the resulting jeopardy to the patient's social and economic status, must be taken into account in assessing disability

For the physician, the medicolegal aspects of a head injury are well worth bearing in mind not only when a case is in litigation, but from the very beginning of an accident, since any case may become the subject of litigation For this reason, the most detailed description of the findings at the first examination may be very useful later

Examination

Scalp

All scalp wounds must be carefully described Some patients are first seen in an admitting room after a scalp wound has been sutured, but in all cases it is advisable to note the type of wound found, personally or by a member of the emergency staff This may provide valuable information about the nature of the injuring object, should the case come to litigation

Skull

Just as the scalp wound may suggest the nature of the injuring object, the pattern of skull deformation and the resultant fracture may give important information about the site of impact.[4] However, in certain cases, it may be difficult or impossible to tell, except in a general manner, the site or sites of impact which resulted in the fractures.

Intracranial Contents

An accurate evaluation and description of the dural and cerebral injuries are essential in all cases of open head injury Photographs may later be valuable should need arise to describe the extent of the damage Any tissue removed at operation should be sent to the pathologic laboratory for study the pathologist s finding that tissue has been destroyed is undisputed evidence of intracranial injury Such laboratory study may also help to estimate the lapse of time between injury and operation, particularly in the case of chronic subdural hematoma. Microscopic study of fibroglial scars cystic lesions arachnoidal proliferation, abscess wall or hematoma capsule may reveal the approximate time it had taken for the lesions to develop. While extradural and subdural hematomas are frequently of traumatic origin, a skull fracture may have been suffered when the patient fell as a result of a sudden stroke the history the circumstances, a description of the fall, and the place where the patient was found may all be important factors if an opinion has to be given about the case later In some cases, alcoholism misleading neurologic symptoms, or the plaintiff's advanced age obscure the possible presence of a traumatic intracranial hemorrhage. In such cases, a chronic subdural hematoma may be found at autopsy and careful investigation almost always leads to the discovery that a head injury had occurred several months before the hospital admission

Rupture of a saccular aneurysm or of a vascular malformation may be associated with an accident Stress and strain may affect such

lesions, resulting in states ranging from mild complaints to serious sequelae or death. If prolonged disability occurs, the problem of compensation may arise, as in the case of a fireman who collapsed with fatigue and headache after a long and tiring tour of duty at a fire. He was able to go home, but 4 days later was admitted to the hospital where he died shortly thereafter; autopsy revealed a ruptured congenital aneurysm. It was contended successfully that his death was the result of an accident and that his family was entitled to benefits.

Intracranial vascular lesions may become manifest some time after a head injury — the so-called delayed traumatic cerebral hemorrhage. The patient seems to recover from the head injury, then days or weeks later succumbs to a cerebral hemorrhage associated with paralysis. The signs and symptoms simulate the hemorrhage of apoplexy or stroke. When such an intracerebral hemorrhage occurs soon after a serious blow to the head, particularly in the old, the blow may well be the provocative mechanism, but a lapse of more than 4 weeks and minor head injury exclude the likelihood of this relation.

Late Apoplexy

The so-called late apoplexy,[1] characterized by the occurrence of "apoplexy" several weeks after recovery from a head injury, is usually unrelated to the injury. In many such cases the cause of the stroke is found to be thrombosis of a major blood vessel supplying the brain. If the interval between the head injury and the thrombosis is several weeks to 2 or 3 months, they are not related. This is particularly true in those cases in which the head injury was minor and the patient returned to work and continued at work for several weeks before the sudden onset of paralysis.

Such is not the case when the paralysis appears soon after injury, and is due to thrombosis of the internal carotid artery or other major vessel. The probable cause is then a contusion of the vascular intima caused by hyperextension, by kinking, or by an actual blow to the vessel by a fracture deformation.[4, 5] In these patients the thrombosis

becomes complete within several hours or within 24 to 48 hours, and focal signs usually become manifest.

Occasionally, hemorrhage may result from rupture of blood vessels running across a cystic area which is a residual of an earlier injury The cyst may contain clear or yellow fluid and be crossed by bands of tissue, some of them consisting of blood vessels. Rupture of these vessels may produce the so-called late apoplexy We have seen such a hemorrhage in a 16 year old patient with a porencephalic cyst due to a birth injury

A vascular lesion which creates a communication between the carotid artery and the cavernous sinus is usually of traumatic origin In most cases, the symptoms and signs develop rapidly and become evident within hours to a few days, but in a few, in whom the arteriovenous communication is small, several months may pass before the diagnosis is finally made. During this period, the patient's only complaint may be of a noise in the head, in a routine examination, this symptom may not be evaluated properly

Delayed Inflammatory Complications

Inflammatory cerebral disease may occur some time after a head injury and in some cases constitute a medicolegal problem Meningitis may occur long after a fracture through the frontal sinus and cribriform plate area, a fracture which may create a cranionasal fistula. If skull roentgenograms some time after the injury fail to reveal the fracture, which has healed in the interval, or the patient has not realized that the fluid draining into his nose, throat, or ear is cerebrospinal fluid or if the injury had seemingly been a mild one, the cause of the meningitis may be hard or practically impossible to establish correctly

So, too, may the onset of a brain abscess be both delayed and insidious, and not always associated with meningitis. In some cases, the clinical findings are more typical of a brain tumor, particularly when the abscess has been present for weeks or months. But no matter how

long after a head injury, an abscess may be the direct result of the injury.

Another late complication of head injury is an osteomyelitis of the skull A swollen area of scalp, which on examination is found to be a collection of pus with destruction of the underlying bone, may have been caused by an injury several weeks or months earlier; or, what appears to be a minor scalp laceration may heal satisfactorily, only to begin draining many weeks later because of the underlying contaminated material

All such cases may have medicolegal implications Careful scrutiny and study of the complete history of a case permit its evaluation in a manner fair to both plaintiff and defendant

Relation Between Head Injury and Cerebral Diseases

A head injury may be a factor in causing or accelerating an intracranial disease process, particularly of brain tumors With the possible exception of benign meningiomas,[2] which may occur at sites of skull injury, brain tumors are not caused by trauma But injury may aggravate the condition of a brain tumor The glioblastoma, because of its vascularity, bleeds easily, hemorrhage of a glioblastoma may occur spontaneously, or as a result of a head injury A typical case is that of a man well enough to work who falls and strikes his head He may advance various reasons for his fall, often overlooking or ignoring the bout of dizziness that caused him to lose his balance The disabling, persistent headaches and the signs of serious cerebral involvement which eventually develop, leading to neurologic studies and finally to operation, may then reveal the glioblastoma, with evidence of hemorrhage, or some other brain tumor In other cases, the tumor may have been present at the time of a head injury without having caused any symptoms If the head injury is severe, and associated with increased intracranial pressure and cerebral edema, it may have an

adverse effect on the tumor, eventually, the presence of the tumor may be suspected leading to operation and microscopic study to establish its type In such cases, the relation between the head injury and the accelerated development of the tumor may be satisfactorily established in court In still other cases, the patient returns to work after a head injury and performs satisfactorily for several weeks. Then he begins to complain increasingly of symptoms referable to the head, and eventually a brain tumor is diagnosed and its presence proved by operation. It is unlikely in such a case that the head injury was an aggravating factor, particularly if the tumor is malignant. The course of the tumor and the eventual death were not affected by the head injury

Many other central nervous system diseases may be ascribed to head injury, but in our experience no definite relation has ever been established between such conditions and head injury

A thorough appraisal of all the clinical and pathologic elements in the individual case usually enables the examiner to make an accurate, fair estimate of the cause-and-effect relation The pathologic features found on operation or at autopsy are of the utmost importance. The accuracy of the latter depend on the quality of the coroner s pathologic laboratory The nonpolitical medical examiners departments of pathology, which have replaced the outdated elective coroner system in the larger cities, have made many important contributions to the medicolegal aspects of head injuries Since a competent pathologist is usually a member of the staff of most hospitals of 150 beds or more, many of the so-called coroner s cases may be examined by the pathologist when death occurs in the hospital. The surest means of resolving conflicting evidence in medicolegal cases is by obtaining the closest cooperation of all medical facilities — clinical, surgical, and pathologic.

Court Presentation

It is noteworthy that only 2 to 4 Workmen's Compensation cases out of every 100 require medical testimony before the Commissioner or his deputy Usually, the cases are settled without a hearing, on the basis of medical reports acceptable to both parties, as represented by their attorneys

The presence of the medical examiner who carried out the examination at the request of the plaintiff's or defendant's attorney may be required at a hearing The subpoena requiring his attendance commonly allows arrangements to suit his convenience The fee for his testimony as an expert varies from \$25 00 to \$100 00, depending on the time involved, as well as on the community and the precedents set

The clinical case report, copies of which are usually already in the possession of both attorneys, is made available by the physician at the hearing Although he may have had the report, an attorney may not understand clearly the relation between the content of the physician's testimony and the arguments which he must use to win the case It is often advisable, therefore, for the examiner to acquaint the attorney, before the hearing opens, with the medical conclusions which will in all likelihood develop from his testimony This may avoid embarrassment, particularly if the testimony hurts rather than helps the attorney's case, since the attorney may not have understood all the details of the medical problem involved

The physician presents his testimony after being sworn in by the Commissioner and identifying himself by name and as an expert by virtue of his training and experience The manner in which he gives his testimony can determine the efficiency and clarity of the hearing Attorneys in court sometimes neglect the simple courtesies which they would scrupulously observe everywhere else This technic may hurt the pride or feelings of the medical witness during cross examination by the opposing attorney. He can best protect himself by stating the

facts as determined by his examination in an unperturbed, objective manner His answers must not be motivated by anger flippancy or duplicity His only function is to describe the patient's clinical state and to present his evaluation of the alleged injury in terms of the disability which he found At times, a hypothetic question or problem is advanced for his opinion, such a question should be considered on its own merits, without regard to the case being tried Equanimity in answering questions simply, concise statements, a basic understanding of legal form and of attorneys methods and a broad knowledge of the problem involved can turn a medicolegal hearing or trial into an interesting rather than a distressing experience for the physician

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Chapter XIII

GENERAL CARE AND THE MANAGEMENT OF NONSURGICAL CONDITIONS

The conservative management of patients with craniocerebral trauma is determined by the degree of the injury With a minimal injury, the patient is observed in the hospital for 2 or 3 days, thereafter, he is seen from time to time, since a subdural hematoma or hygroma[10] can develop in some cases of minor injury

The patient with a moderately severe injury usually shows some improvement in the course of several hours to several days Small scalp lacerations are repaired, and the patient is then made comfortable with mild sedation, and an ice bag applied to the head The bed should be in a semi-Fowler position The pulse, blood pressure, respiration, and temperature should be checked at intervals A careful neurologic examination should be done while the patient is in the hospital, and neurologic observation continued during the hospital stay so as to detect the development of complications which might require surgical intervention

Not many patients with a serious head injury need immediate surgical treatment, but all require personalized unremitting care This is the first essential, and there can be no doubt that such care, and a



Fig 82. Oxygen administration in management of unconscious patients with head injury (*a*) Patient in oxygen tent, an inadvisable method (*b*) Administration by tube and patient exposed to air for temperature control second tube in position, extending into stomach for feeding closer view of jar and stand intravenous feeding (b^2) (*c*) Administration to tracheotomized patient.

tracheotomy when indicated, have been lifesaving in many cases, even in some near death on hospital admission The nursing must be meticulous and constant Frequent turning of the patient and bladder care are important It is essential to keep the air passages clear and patent, and prevent the aspiration of vomitus by washing out the stomach if necessary An adequate intake of food and fluid should be assured, if necessary by nasal catheter extending into the stomach (Fig 82*b*).

Respiratory embarrassment may be improved by careful and continuous suction of the upper respiratory passages But continued respiratory distress must be relieved by tracheotomy (Fig 82*c-d*), which usually can be performed at the bedside

Technic of Tracheotomy[2, 14] (Fig. 83)

In a thin, long-necked individual with a small thyroid gland, tracheotomy is a simple procedure, whereas in an obese individual with a short thick neck, tracheotomy, or even intubation, may be difficult The scars of previous operations on the neck, such as subtotal or total thyroidectomy, increase the difficulty of the procedure and prolong the time required to provide an adequate airway The indications for tracheotomy are. labored breathing, cyanosis, a rising temperature, a weak rapid pulse, and retraction of the intercostal muscles Before proceeding with a tracheotomy, however, the patient's chin should be elevated and the tongue pulled forward, to make sure that this is not the cause of an obstructed airway A suction apparatus should be at hand before starting the tracheotomy.

The head and neck should be extended over a rolled sheet or pillow (Fig. 83*a*), so as to obtain maximum exposure of the trachea and bring it forward toward the neck surface The neck is made aseptic with iodine and alcohol, and sterile towels are placed about it A transverse incision is made from the inner border of one sternocleidomastoid muscle to the inner border of the other, about 1½ inches below the upper border of the thyroid cartilage. The subcutaneous

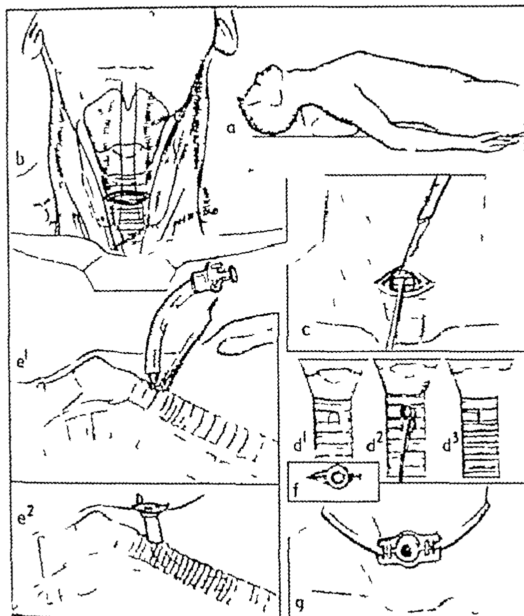


Fig. 83 Technic of tracheotomy (*a*) Hyperextension of neck over pillow (*b*) Pertinent anatomy of neck including veins on both sides of trachea in operative area. (*c*) Trachea grasped by 2 instruments and elevated slightly (*d*) Semicircular or vertical incision of second tracheal ring (*e*) Insertion of tracheotomy tube. (*f*) Suturing of wound. (*g*) Tracheotomy tube in place with attached tape tied around neck.

tissues can usually be spread with blunt scissors away from the midline without injuring vascular channels The enveloping fascia of the neck muscles is split longitudinally in the midline, and the trachea is located and freed from the surrounding fascia with a hemostat and blunt scissors (Fig 83*c*) It is then grasped with double-toothed instruments (thumb forceps or hemostats) and elevated slightly while a semicircular or vertical incision is made with a sharp knife in its second cartilaginous ring (Fig 83*d*), the incision should be large enough to take the longest and largest tracheotomy tube or cannula in the set for adults With the forceps pulling the trachea toward the skin incision, the tracheotomy tube is inserted (Fig 83*e*) Any mucus expelled during the coughing spell precipitated by the insertion of the tube should be promptly aspirated by suction, all secretions in the tracheobronchial tree are removed by suction through a soft, small rubber catheter inserted through the tube down the trachea, care being taken that the flow of air is not impeded The incision is closed by a few subcuticular silk sutures on either side of the tube and one or two skin stitches (Fig 83*f*) The tube is held in place by tying the attached tapes around the neck (Fig 83*g*).

Some surgeons prefer the vertical incision (Fig 83*d'*) because of the slighter likelihood of tracheal stenosis with this incision The incision should not be closed tightly, since emphysema and pneumothorax may occur if there is leakage around the loosely applied tube. Because of this possibility, some surgeons prefer not to suture the wound When performing a tracheotomy in a child or an infant, it should be kept in mind that the trachea is near the carotid artery and jugular vein

Whatever the means, keeping the respiratory passages clear is even more essential than the administration of oxygen The tracheotomy tube should be of the largest possible size so as not to diminish the vital capacity, and it must be kept clear by continuous suction. Intratracheal intubation with a methyl methacrylate (Lucite, Plexiglas) tube can be used in some cases, particularly for short periods of a day

or two Although such tubes have remained in the trachea for several days without ill effect, erosion of the tracheal wall is possible. A closed tracheotomy method which may simplify the problem of performing a tracheotomy in a desperately ill patient has recently been described by Shelden and co-workers[13]

It is important in these cases that the stomach contents be removed by a Levine tube in order to prevent the aspiration of vomitus.

Oxygen Administration

For the administration of oxygen (Fig 82) it is best not to use a tent, the reasons being (*1*) The tent may be construed as a form of treatment by the attendants and the patient therefore be left unobserved for too long an interval (*2*) Sheets and blankets covering the patient tend to keep his temperature up (*3*) A patient in a tent is less accessible for the necessary frequent cleaning of the respiratory passages. A better way to administer oxygen is through a tube in the posterior nasal pharynx, or, in the presence of a tracheotomy by means of a small catheter extending toward the tracheotomy tube, a large rubber tube should not be used for this purpose, since it reduces the vital capacity

Temperature Control

A simple way to reduce body heat is to expose large areas of the body surface and thus increase evaporation. Ice bags sponge baths and ice water enemas if the temperature is over 104 F, all help to control high temperatures Hypothermia has recently come into use in cases of serious head injury This drastic decrease in temperature helps to reduce metabolic requirements so that hypoxic levels are better tolerated by the patient. Hypothermia may be induced by surrounding the patient with ice filled plastic bags, or as suggested by Lewis and associates,[7] by means of a cooling fluid in a blanket covering the patient. Promazine (Sparine) in adequate dosage should be administered during the hypothermic interval. A patient kept at hypothermic levels

should be examined frequently, to avoid overlooking the development of a mass lesion, for which hypothermia is of no help

Hypothermic levels of 31 to 35 C have recently been used in the management of patients with signs of brain stem involvement In some patients the hypothermia not only helped to improve the general condition but also eliminated the frequent attacks of decerebrate rigidity which occur with this type of injury Several investigators have reported a definite improvement in the mortality rates of severely injured patients when treated with hypothermia[7,9]

Food and Fluids

For intranasal feeding, 2,000 calories in 2,500 cc of fluid per day for the adult and proportionately smaller amounts for the younger age groups may be used Additional fluid, when needed, may be administered parenterally

Use of a solution which is more isotonic with the tissues and the intercellular fluid than saline solution has recently been suggested, it contains 2 5 per cent glucose and 0 42 per cent sodium chloride.[1] We have had no experience with this solution, but it may be of value in a patient with a profound injury. On the other hand, some studies on rising intracranial pressure seem to indicate that pressures as high as the diastolic blood pressure do not necessarily affect vital or cerebral functions.[1, 12] Dehydration, a method in common use some 25 years ago, is no longer advocated

Blood Chemistry

Urinary excretion of sugar, sodium, potassium, chlorides, and nitr[illegible] enous products, and the blood levels of urea nitrogen, s[illegible] tassium, chlorides, and glucose, should be determined i[illegible] remains unconscious for several days Metabolic distur[illegible] tral nervous system origin, which may be more comm[illegible] ally believed, can thus be detected Lowered sodium levels should be corrected gradually (over several days)

administration of adequate amounts of the electrolytes in solution in the form of saline solution Hartmann s solution, or potassium chloride solution, as may be indicated In the presence of hyperosmolarity, the patient s renal function and his diet need careful evaluation, the diet may be too high in protein, resulting in deranged kidney function not only for protein, but also for sodium. Decreasing the amount of protein and increasing the carbohydrates and fat may correct the hyperosmolarity in such a case. If, on the other hand, the condition is caused by involvement of the hypothalamus or of the frontobasilar portions of the brain, mercurial diuretics and forced fluids are indicated

A patient with a head injury may have a renal shutdown as a result of extensive injuries elsewhere in the body, the fluid intake in such cases should be restricted during the period of dysfunction

Convulsions

Convulsions in serious head injuries may, in some cases, be initiated by hypoxia. In such cases, relief of respiratory embarrassment by clearing the air passages by suction, by introducing an intratracheal tube, or by tracheotomy, may help to control the seizures. Intensive medical measures are also indicated in the presence of frequent seizures, if anticonvulsant therapy is inadequate, as it may be, Pentothal must be administered intravenously until the seizures stop Open ether anesthesia may be helpful Administration of oxygen during anesthesia therapy is of value Anticonvulsants may be indicated during the convalescent period of these patients.

Lumbar Puncture

The therapeutic value of lumbar puncture to lower cerebrospinal fluid pressure and to remove bloody fluid is questionable, although in some cases of localized cerebral edema decreasing the pressure by this means may be beneficial Rarely, an aphasic patient recovers his speech and an unconscious patient improves immediately after a lumbar puncture. But sudden death especially in children, may also occur

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Use of a solution which is more isotonic with the tissues and the intercellular fluid than saline solution has recently been suggested; it contains 2.5 per cent glucose and 0.42 per cent sodium chloride.[1] We have had no experience with this solution, but it may be of value in a patient with a profound injury. On the other hand, some studies on rising intracranial pressure seem to indicate that pressures as high as the diastolic blood pressure do not necessarily affect vital or cerebral functions.[3, 12] Dehydration, a method in common use some 25 years ago, is no longer advocated.

Blood Chemistry

Urinary excretion of sugar, sodium, potassium, chlorides, and nitrogenous products, and the blood levels of urea nitrogen, sodium, potassium, chlorides, and glucose, should be determined if the patient remains unconscious for several days. Metabolic disturbances of central nervous system origin, which may be more common than is usually believed, can thus be detected. Lowered sodium or potassium levels should be corrected gradually (over several days) by intravenous

administration of adequate amounts of the electrolytes in solution, in the form of saline solution, Hartmann s solution, or potassium chloride solution, as may be indicated In the presence of hyperosmolarity, the patient's renal function and his diet need careful evaluation, the diet may be too high in protein, resulting in deranged kidney function not only for protein, but also for sodium. Decreasing the amount of protein and increasing the carbohydrates and fat may correct the hyperosmolarity in such a case. If, on the other hand, the condition is caused by involvement of the hypothalamus or of the frontobasilar portions of the brain, mercurial diuretics and forced fluids are indicated

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immediately after the procedure Moreover, lumbar puncture may upset the intracranial hydrodynamics and thereby cause increase of a hemorrhage, or, in the presence of a hematoma, increase the risk of incisural herniation Extradural and subdural hematomas are therefore contraindications to the use of therapeutic lumbar puncture A too radical lowering of the pressure by lumbar puncture is inadvisable, a good rule is to bring the pressure down by 25 per cent, even though this may still leave the pressure at a high level The limitations of therapeutic lumbar puncture do not apply to its use for diagnostic purposes It may be used whenever necessary during the treatment of a head injury, and should be used promptly if infection is suspected

Drug Therapy

A number of drugs, among them caffeine sodiobenzoate, atropine, and cortisone, help to combat the effects of head injury Caffeine sodiobenzoate was first suggested for the relief of increased intracranial pressure,[6] but there has been no wide acceptance of its efficacy, nor has its value in relieving cerebral edema been definitely proved. Excellent results have been reported from the use of atropine, 0 1 grain every 6 hours for 2 or 3 days, in adults with severe, closed head injuries[11, 15] However, we have not found this drug to be particularly effective On the other hand, cortisone, starting with 100 mg, with decreasing doses, 3 times a day for 2 days, or prednisone (Meticorten), starting with 20 mg, with decreasing doses, 3 times a day for 3 to 5 days, has given good results Some of our moribund patients improved spectacularly, and patients unconscious for several days, but without intracranial mass lesions, seemed to become ambulatory more quickly than patients not given cortisone Possibly, the beneficial effect of cortisone in head injury is merely its effect in overcoming the stress of a serious injury and the concomitant pathologic process Whatever the drug used, care must be taken not to depend on it to such an extent as to cause the presence of a mass lesion to be overlooked

The formerly used hypertonic solutions are no longer highly re-

garded as a means of reducing intracranial pressure. Although they do cause a drop in pressure and some improvement in the patient's condition, the effect is only temporary as the pressure rises again, the patient once more becomes restless and stuporous. However, 150 to 300 cc. of a 30 per cent sucrose solution, which causes a sustained lowering of the pressure, may be used in selected cases. Urea solution intravenously or by stomach tube, has been reported to be of value in controlling an increase in pressure, without evidence of a rebound after the fall[3]

While proper care will reduce restlessness and thereby minimize the need for sedatives and narcotics, some patients nevertheless need sedation. Codeine and phenobarbital, by mouth or intramuscularly are useful in most cases, unlike morphine they have no effect on the conscious state or on the localizing signs of a dynamic lesion Morphine should not be given, except to a patient with a fracture of a long bone in considerable pain and whose craniocerebral injury is mild Morphine sulfate causes an unmistakable increase in cerebrospinal fluid pressure in severe injury, but in moderate or minimal injuries no adverse affect has been noted[4]

Pitressin-in-oil, intramuscularly or snuff prepared from the dried posterior lobe of the pituitary may be used to combat the polyuria of diabetes insipidus.

Treatment of Some Posttraumatic Conditions

Cerebrospinal Fluid or Bloody Otorrhea and Rhinorrhea

A discharge of clear or bloody fluid from the ear usually subsides spontaneously Only rarely is surgical intervention required generally in cases in which there is a discharge of cerebral tissue as well as fluid

Bleeding from the ear stops spontaneously in the course of 1 to 5 days, as a rule. A semi Fowler position so long as the patient remains in bed is advisable. The ear should be thoroughly cleansed and fluffy gauze applied to the external ear and held in place by a bandage.

Aural bleeding is seldom severe, occasionally, however, hemorrhage from a torn lateral sinus or carotid artery through the external ear may be fatal if the source of the bleeding is not discovered quickly and the bleeding stopped with dispatch Sulfonamide and antibiotic prophylaxis is indicated in all cases of otorrhea For dosages, *see* below

Such prophylaxis should also be used in the presence of cerebrospinal fluid *rhinorrhea*

Meningitis

The best treatment of posttraumatic meningitis is its prevention by means of chemotherapeutic and antibiotic prophylactic therapy, 400,000 to 800,000 units of penicillin and 4 Gm of sulfisoxazole (Gantrisin) daily, used routinely in open depressed fractures and in every case with cranionasal or cranioaural (as indicated by rhinorrhea or otorrhea) fistula, will prevent the occurrence of meningitis in most cases

Should meningitis occur despite such prophylaxis, the dosage of penicillin is increased to 10,000,000 to 15,000,000 units in aqueous solution intramuscularly, or 25,000,000 to 34,000,000 units in 3,000 cc of 5 per cent glucose solution intravenously, daily until the infection is under control, and for 4 or 5 days longer Administration of 2 grains of sulfadiazine per pound of body weight per day is also continued

As soon as the diagnosis is established, the invading organism is identified by smear and culture studies, and its drug sensitivity determined. Therapy can then be altered as the results of these studies become available Once the fever and leukocytosis have subsided, the cerebrospinal fluid should contain few or no pus cells

The treatment just outlined resulted in cure in 7 of 8 recent cases of posttraumatic meningitis at the Grace Hospital and the Detroit Receiving Hospital

Fat Embolism

Not all cases of fat embolism are critical and many may be treated conservatively Supportive measures are of value. These include careful

positioning to provide a good airway, and a tracheotomy, if necessary Cardiac difficulties call for the proper therapy for their control, as do convulsive seizures, in the latter case general anesthesia for the duration of an attack may be used if the anticonvulsants are not effective. If the focal signs seem to indicate a mass lesion it is wiser to assume its possible presence and establish the diagnosis by exploration than to rely solely on conservative treatment.

Postconcussion and Postsubconcussion Syndromes

The management of both syndromes includes reassurance, the use of sedatives, early ambulation, and return to normal activity Headaches may be localized in contused and lacerated areas, local infiltration of Xylocaine hydrochloride may help some of the patients. Drugs for the control of local or general headaches and dizzy spells, and occasionally hypnotics for sleep, may be indicated Manifest interest in the patient's problems helps to reassure him. Careful evaluation of the disability by electroencephalography, and consultations with an ophthalmologist and an otolaryngologist, may suggest more fruitful treatment than continued minimizing of the patient's complaints. If the results of all examinations are negative, the physician may discuss with the patient in a frank manner his problems and their evaluation.

Workmen's Compensation or insurance considerations, or an inadequate pretraumatic personality, may sometimes complicate the picture and lengthen the period of disability In such cases, early settlement may be medically valuable in helping to terminate most of the complaints. A well-organized rehabilitation technic is of great value in reassuring the patient and in helping him to return to full activity

Whiplash (Hyperextension, Hyperflexion) Injuries

Management of whiplash injuries is governed by the type and severity of the injury For the injury consisting of muscle and ligament strains, temporary immobilization, mild heat, massage, and analgesics suffice.

Stretching and tearing injuries of muscles and ligaments probably require closer observation, more intensive care, and more potent analgesia. Temporary immobilization may be helpful, followed by gradually increasing halter neck traction. Continuous halter traction in bed may be beneficial in some cases. This, carefully supervised by a physiotherapist, may also help the patient with an intervertebral disk lesion or cervical nerve injury. Self-treatment may later be continued at home.

Fractures and fracture dislocations of the cervical spine are treated by conventional methods, including immobilization and the use of skull tongs for traction, if indicated.

"Chiropractic" manipulations of the neck should be avoided. Although such manipulations sometimes give immediate relief, it is only temporary and the pain soon returns. They are a source of danger, since they may damage an intervertebral disk, and sometimes cause spinal cord injury.

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Chapter XIV

SURGICAL MANAGEMENT AND TECHNICS[29]

About 12 per cent of patients with acute head injury need surgical treatment An additional 5 to 8 per cent may need operative intervention for the diagnosis of posttraumatic complications or for the repair of skull defects

The interpretation of signs and symptoms as indications for possible surgical intervention must be cautious No routine decision is adequate, and every case must be individually evaluated By repeated assessment of the patient's condition, one is usually able to decide about the need for surgery within a reasonable time Good surgical judgment is only possible on the basis of constant observation of the patient.

The time to operate is generally as soon as the patient's condition permits Skull fractures necessitating intervention are treated immediately if the patient is conscious and has a normal blood pressure or if his condition is stable In the case of mass lesions, exploration or intervention is instituted with or without preliminary diagnostic operative procedures

General anesthesia with intratracheal intubation, local anesthesia in some cases, and a combination of local anesthesia and Pentothal intra-

venously are used in our clinic Block anesthesia by infiltration of the supraorbital and supratrochlear nerves and the branches of the auriculotemporal nerve near the ear gives a large anesthetized area involving the frontal and the anterior parietal regions For angiography and ventriculography, local infiltration is preferable Blood and plasma are used as need arises. The preparation of an infant or child for surgery should include cannulation of a vein, so that a transfusion can be given without delay should it become necessary

Antibiotics are routinely used in cases with open wounds of the head and also postoperatively after exploratory surgery In the presence of proved meningitis, effective dosages of antibiotic drugs are used for 8 to 12 days, or as long as may be necessary to control the infection, we have used as much as 5,000,000 to 12,000,000 units of penicillin intramuscularly, or 36,000,000 units in 3,000 cc. of 5 per cent glucose intravenously with 2 grains of sodium sulfadiazine per pound of body weight per day As soon as the type of infection has been determined and the drug sensitivity of the organism(s) has been established the proper medication is instituted. Combinations of sulfadiazine and drugs of the tetracycline group, as well as penicillin, have been frequently used. When drugs are indicated for the prevention of infections, 1,000,000 units of penicillin and 6 to 8 Gm of Gantrisin per day are administered for 8 days, in the presence of persisting cerebrospinal fluid rhinorrhea or otorrhea therapy is continued until the complication is cleared up One should be ready to revise drug management at any time, as circumstances and the patient s condition dictate.

Postoperative Bleeding

This complication requires the earliest possible diagnosis and treatment. Early exploration may minimize the irreparable damage due to uncal herniation and brain stem compression. Among the signs and symptoms suggesting clot formation are increasing stupor and focal phenomena not present earlier Third nerve paralysis, with increasing hemiparesis and unilateral pupillary dilatation in a semi

conscious patient who is becoming more stuporous, are important signs The pulse rate and blood pressure should be noted every 30 minutes in the immediate postoperative period, they may suggest the presence of a complication by revealing a bradycardia or a rise in blood pressure in a patient who is growing more, rather than less, stuporous

Occasionally, a patient whose subdural hematoma has been satisfactorily evacuated may relapse into increasing stupor Re-exploration at the site of the hematoma may be negative, but more posterior contralateral exploration may reveal a posteriorly located hematoma previously missed

It is our practice to obtain a bilateral carotid angiogram before proceeding with re-exploration in patients in whom postoperative bleeding is suspected The angiogram may show the outlines of a unilateral or bilateral collection of blood

Scalp Lacerations (Figs. 84-85)

Careful cleansing and debridement are essential for all scalp lacerations from the simplest to the most severe, the degree of debridement obviously being governed by the type and severity of the laceration Closure is accomplished by sutures in 2 to 4 layers, depending on the site of the laceration When it is over muscular tissue, as in the temple area, a 4 layer closure is indicated (*1*) suture of the muscle, (2) suture of its overlying fascia, (*3*) suture of the subcutaneous tissue, and (*4*) suture of the skin For a laceration over the vertex or the frontal area, a 2 layer closure usually suffices

Single Lacerations

Repair of a single laceration is accomplished by excising or freshening the edges of the cut with a sharp knife, and closing in 2 layers

The lacerations caused by *low-velocity* missiles are treated like any other simple scalp laceration Since the tissue around the laceration is not extensively devitalized, it is sufficient to freshen the edges by de-

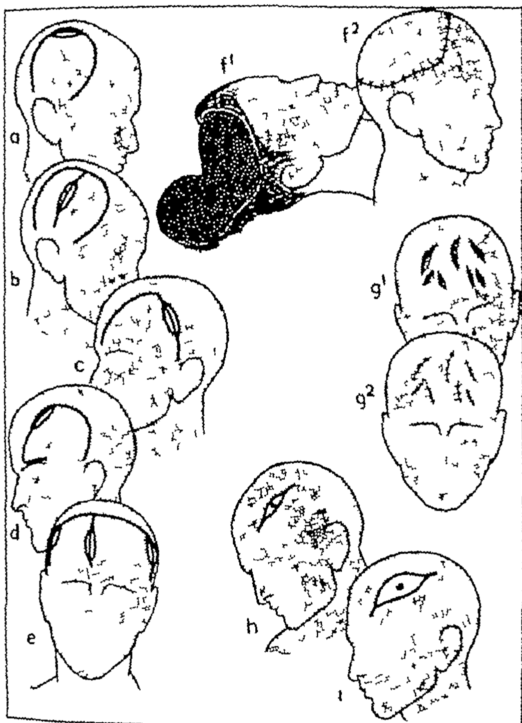


Fig 84 Repair of scalp lacerations. (*a–e*) Flap method of excising lacerated scalp tissue in penetrating injuries (*f*) Avulsed scalp and its repair (*g*) Multiple lacerations and their repair (*h*) Excision for simple laceration (*i*) Excision for high-velocity penetrating injury

bridement, taking care to save as much of the scalp tissue as possible In penetrating injuries caused by *high-velocity* missiles, considerable tissue is devitalized about the wound of entry Wide debridement is therefore necessary, in order to excise all the devitalized tissue (Fig 84*i*). If the laceration is extensive, necessitating still wider excision, the flap method of repair is advisable (Fig 84*a-e*) The scalp is mobilized by an appropriate pattern of incision (Fig 85*a-b*), care being taken to save, so far as possible, the blood supply of the region Gaps in the skin are repaired with split-thickness skin grafts, as are defects in the scalp remaining after mobilization of the flap

Multiple Lacerations

Systematic excision of all the edges of the lacerations may entail considerable loss of tissue and result in tight approximation The edges should therefore be trimmed with great care, the wounds cleansed meticulously, with soap and water, if necessary, before using an antiseptic solution, and then closed in 2 layers If there is actual loss of tissue, various types of incision patterns can be used to mobilize the scalp in order to cover the area of loss (Fig 84*g*).

Shearing Injuries

A portion of the scalp may be evulsed (Fig $84f^1$), or sheared off completely The sheared off or evulsed portion is carefully washed, replaced, and sutured in place (Fig $84f^2$) We have found in many such cases that large portions of the evulsed scalp remained vital, even after being used as a free graft

The bone under the lacerated scalp should be inspected by the gloved finger for the presence of fracture, when the pericranium is torn, the skull surface may be visually inspected. In general, the pericranium should not be opened if it is intact In all cases of scalp laceration, skull roentgenography is essential, to determine the possible presence of fractures underlying the laceration or in other parts of the head

When lacerations have not been repaired for several hours to days,

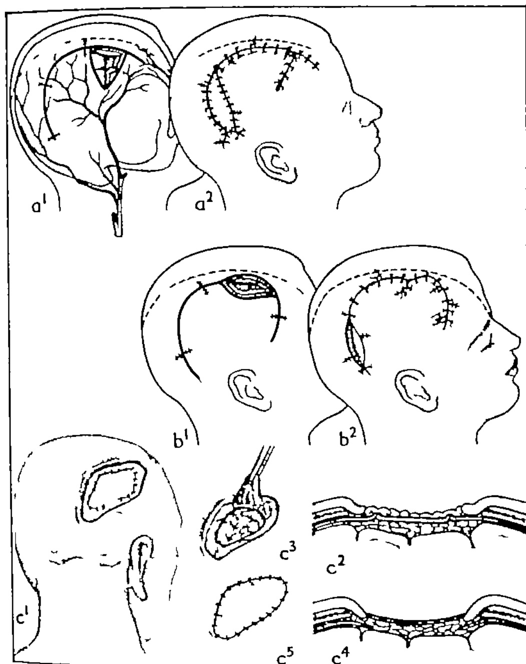


Fig 85 Management of extensive loss of scalp. (*a*) Area of loss, line of excision, and repair gap in posterior portion of skin flap repaired with split-thickness skin graft. (*b*) Repair of scalp loss small defect in posterior portion of flap to be repaired with split-thickness skin graft. (*c*) Repair of dural and scalp defects temporary dural graft (c^1) dural graft covered with granulation tissue (c^2) removal of dural graft (c^3) and split-thickness skin grafts for dural and scalp defects (c^4–c^5)

a primary debridement can be done successfully Often, a delayed primary debridement is successful, with the wound healing completely and by primary intention This is particularly true if antibiotics and sulfonamides are used before and during the management.

In severe head injury, the problem of scalp laceration is part of the larger problem of management of the whole injury When there are underlying pathologic conditions, such as a comminuted fracture, depression, or evident dural tear with brain tissue extruding through the laceration, the skin laceration is repaired in the course of the repair of the entire injury. Under these circumstances, if the patient is very sick, a dressing is applied, and resuscitative measures are used until the patient's condition permits his being taken to the operating room

Linear Fractures (Fig. 86C)

Open linear fractures are inspected for contamination and the presence of foreign matter between the edges of the fracture line If such material is noted, the edges of the fracture are excised An initial opening is made with a burr and a bone rongeur is used for the excision If the fracture looks clean on gross inspection, the wound may be closed after thorough washing and debridement of the scalp laceration. In the presence of linear fracture by high-velocity objects, exploratory trephination to rule out an extradural or subdural hematoma has been suggested by some[17, 38]

Open Depressed Fractures (Figs. 86-87)

The rationale of management of open fractures includes the prevention of infection, the removal of a compli[illegible] hematoma, and measures to minim[illegible] ttraumatic sequ[illegible] erative roentgenography, includ[illegible] opic roentgen[illegible] mportant, providing a clue to the [illegible] mage and th[illegible] d location of bone fragments Po[illegible] ntgenogra[illegible] visable, confirming

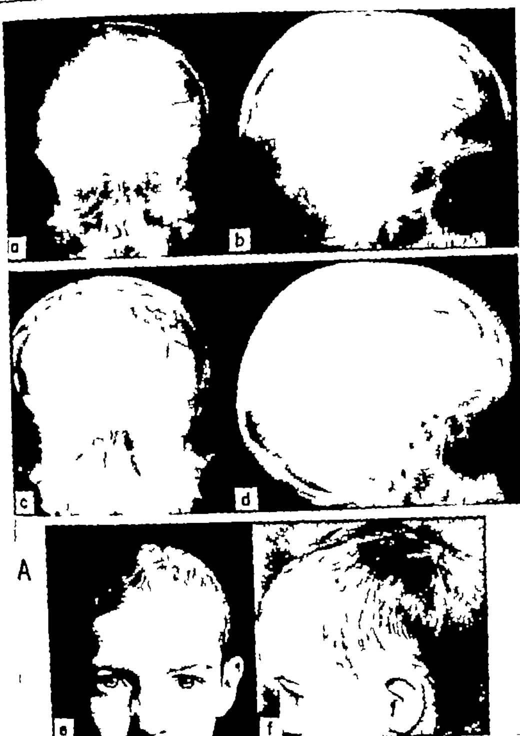


Fig 86. Management of depressed fractures. (A) Simple depressed fracture. (a–d) Pre and postoperative roentgenograms of fracture in left frontal area dura intact and no subdural abnormality circular button of bone removed with trephine, depressed area elevated bone button replaced, and wound closed in layers. (e–f) End result and type of incision used. (*Continued on next page*)

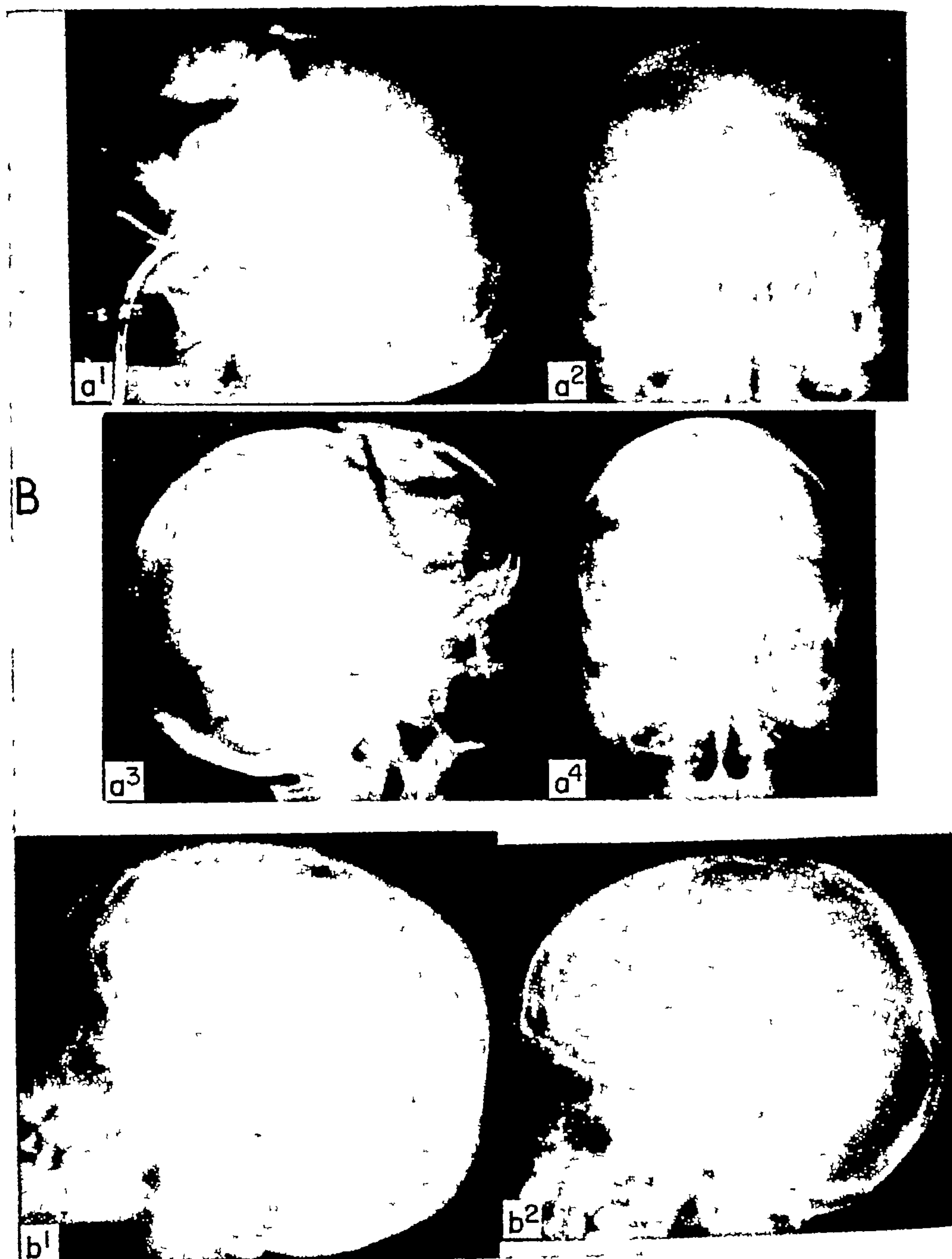


Fig 86 (*continued*). (*B*) Management of open depressed fracture with evulsion (*a*) Before and after repair of fracture caused by crane hook, dura torn, brain macerated, after dural repair, bone fragments replaced and held in place with wire sutures (*b*) Fracture with evulsion of frontal bone by automobile accident, frontal bone replaced and held in place (b^2).

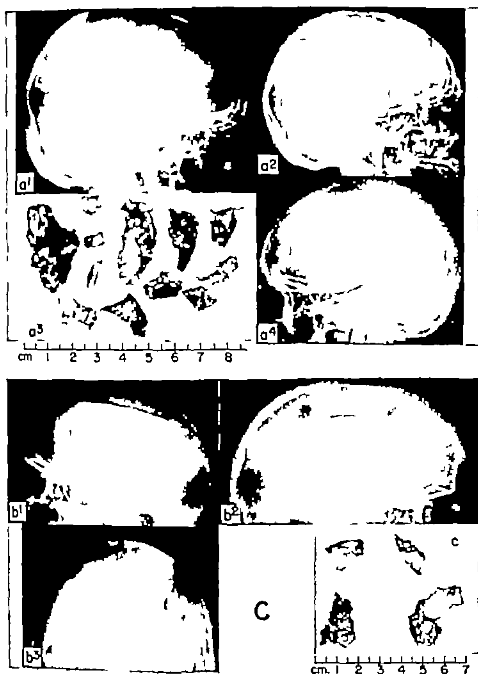


Fig. 86 (*continued*) (*C*) Open depressed fracture of right parietotemporal area. (*a*) (a^{1}) Skull defect (a^{3}) Fragments of bone removed (a^{4}) Tantalum cranioplasty (*b*) Open linear depressed fracture. (b^{1}–b^{3}) Edges excised because of contamination. (*c*) Bone fragments with hair removed in a case of open depressed fracture.

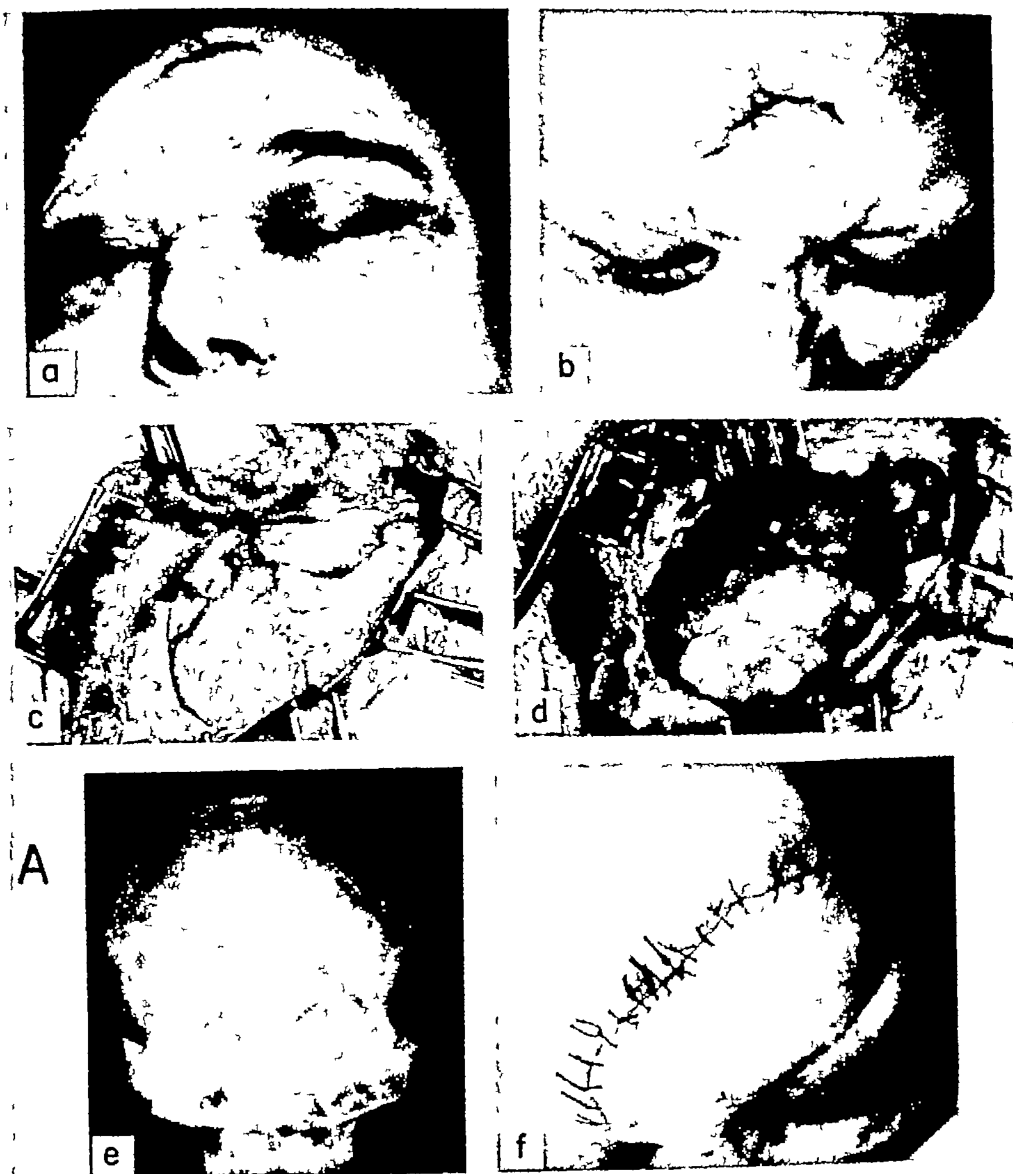


Fig 87 Management of skull fractures (*A*) Open, comminuted fracture of left frontal area (*a–b*) Scalp laceration not in immediate vicinity of fracture (*c*) Site of fracture exposed after excision of lacerated scalp, with cerebral tissue visible between bone fragments (*d*) Removal of bone fragments revealed 2 extensive dural tears (1 extending to roof of fractured frontal sinus), and macerated cerebral tissue, dural tears repaired after excision of macerated tissue (*e*) Preoperative roentgenogram. (*f*) Wound closed in layers, without drainage, skull defect repaired 6 months later by tantalum plate.

B

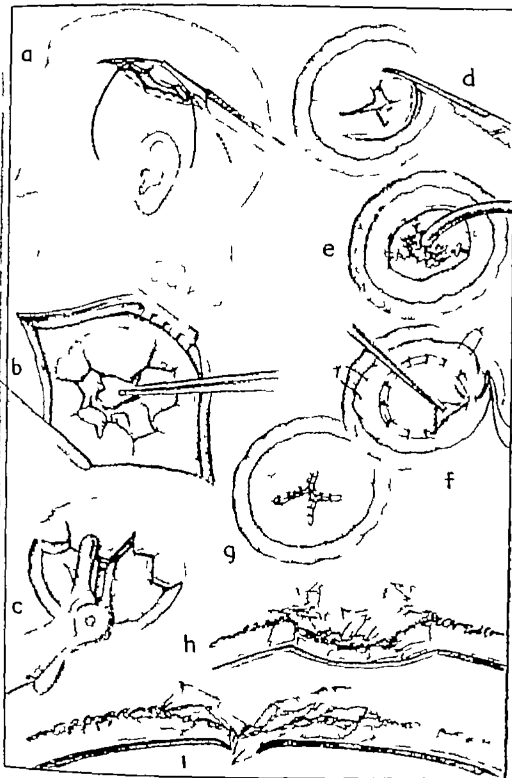


Fig. 87 (*continued*) (*B*) Management of open depressed fracture in left parietotemporal region. (*a*) Injured area exposed. (*b–c*) Bone fragments removed (*d*) Torn dura excised. (*e*) Lacerated cerebral tissue excised by suction. (*f*) Dural tear repaired by fascial graft. (*g*) Dural laceration sutured after debridement (*h–i*) Diagrams of depressed fracture, with and without dural penetration

the removal of all bone fragments An open wound must be inspected carefully; the character of the wound often suggests the most advisable incision to be used so as to provide as much scalp tissue as possible for good closure

The principles of managing an open depressed fracture are exemplified by the following steps for a fracture in the left parieto-temporal area. (*1*) The area is exposed by a skin flap around the ear or by a curved incision along the side of the head (dotted line, Fig. 87*B*, *a*) (2) The fragments of the shattered outer and inner tables thus exposed are carefully removed so as to avoid laceration of the underlying dura (Fig 87*B*, *b-c*) (*3*) Removal of the bone fragments exposes the dura. If a tear is found, the torn portion may have to be excised in circ fashion (Fig 87*B*, *d*), and the defect repaired with a transp temporal fascia or fascia lata (Fig 87*B*, *f*), or it may be p freshen the edges by careful debridement and suture the te 87*B*, *g*) The cerebral surface under the lacerated dura m spected; if laceration or pulping of brain tissue is foun or removal by suction of the pulped tissue (Fig 87*B*, *e*) care being taken to remove only the minimum essential portant to the body economy

Complications such as tears of dural sinuses or arteries, wi hemorrhage, fracture of the frontal or paranasal sinuses, and injury call for special technics

In the young, and particularly in *infants*, a depressed fractu absence of a dural tear can often be managed by elevating the de area This is done by introducing an elevator through a small or burr opening at the border of the depressed area and raisi entire area of depression (Fig. 86*A*)

Depressed Fractures of Frontal Sinus Area (Figs. 88-91)

Surgical treatment of an open depressed fracture of the frontal sinus or of the region around it should be undertaken as soon as the patient condition permits Careful preoperative roentgenography, including

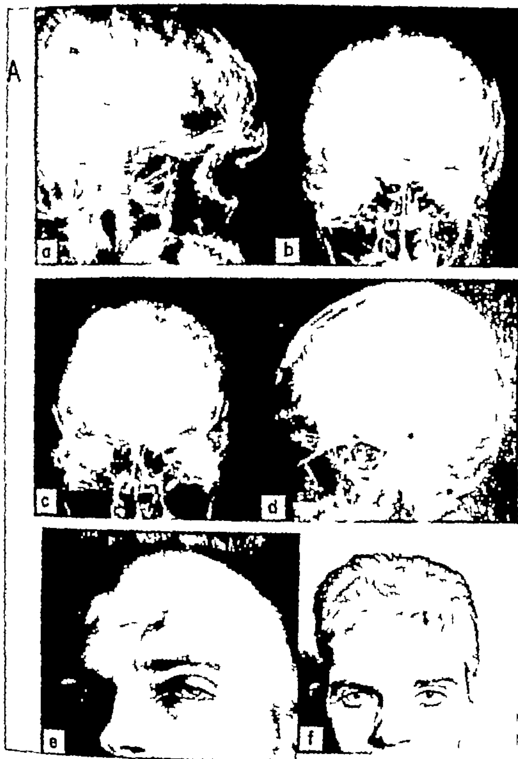


Fig 88. Management of open depressed fracture of frontal sinus area. (*A*) Pre and postoperative roentgenograms (*a–b*) note marked comminution of anterior and posterior walls of frontal sinus and appearance after removal of bone fragments and repair of dural tears skull defect (*e*) repaired 8 months later by osteoperiosteal transplant from outer skull table (*c–d*) (*Continued on next page*)

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Complications such as tears of dural sinuses or arteries, with resultant hemorrhage, fracture of the frontal or paranasal sinuses, and cerebral injury call for special technics

In the young, and particularly in *infants,* a depressed fracture in the absence of a dural tear can often be managed by elevating the depressed area This is done by introducing an elevator through a small trephine or burr opening at the border of the depressed area and raising the entire area of depression (Fig 86*A*)

Depressed Fractures of Frontal Sinus Area (Figs 88-91)

Surgical treatment of an open depressed fracture of the frontal sinus or of the region around it should be undertaken as soon as the patient's condition permits Careful preoperative roentgenography, including

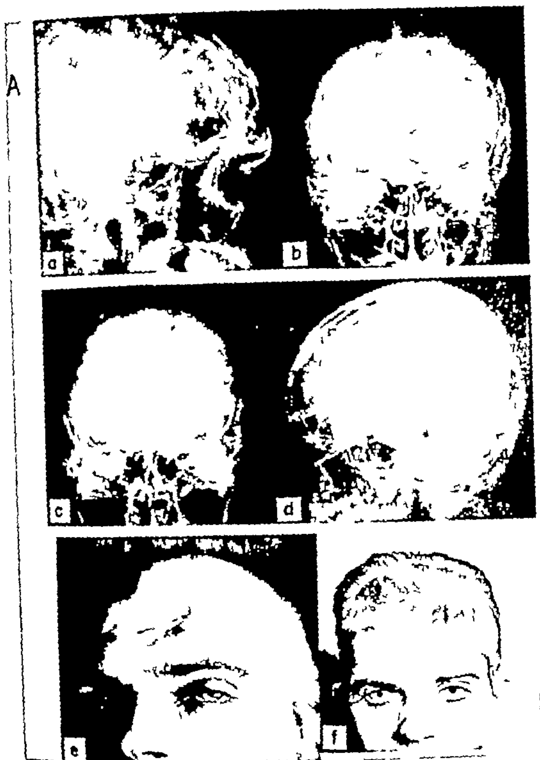


Fig. 88. Management of open depressed fracture of frontal sinus area. (*A*) Pre and postoperative roentgenograms (*a–b*) note marked comminution of anterior and posterior walls of frontal sinus and appearance after removal of bone fragments and repair of dural tears skull defect (*c*) repaired 8 months later by osteoperiosteal transplant from outer skull table (*c–d*) (*Continued on next page*)

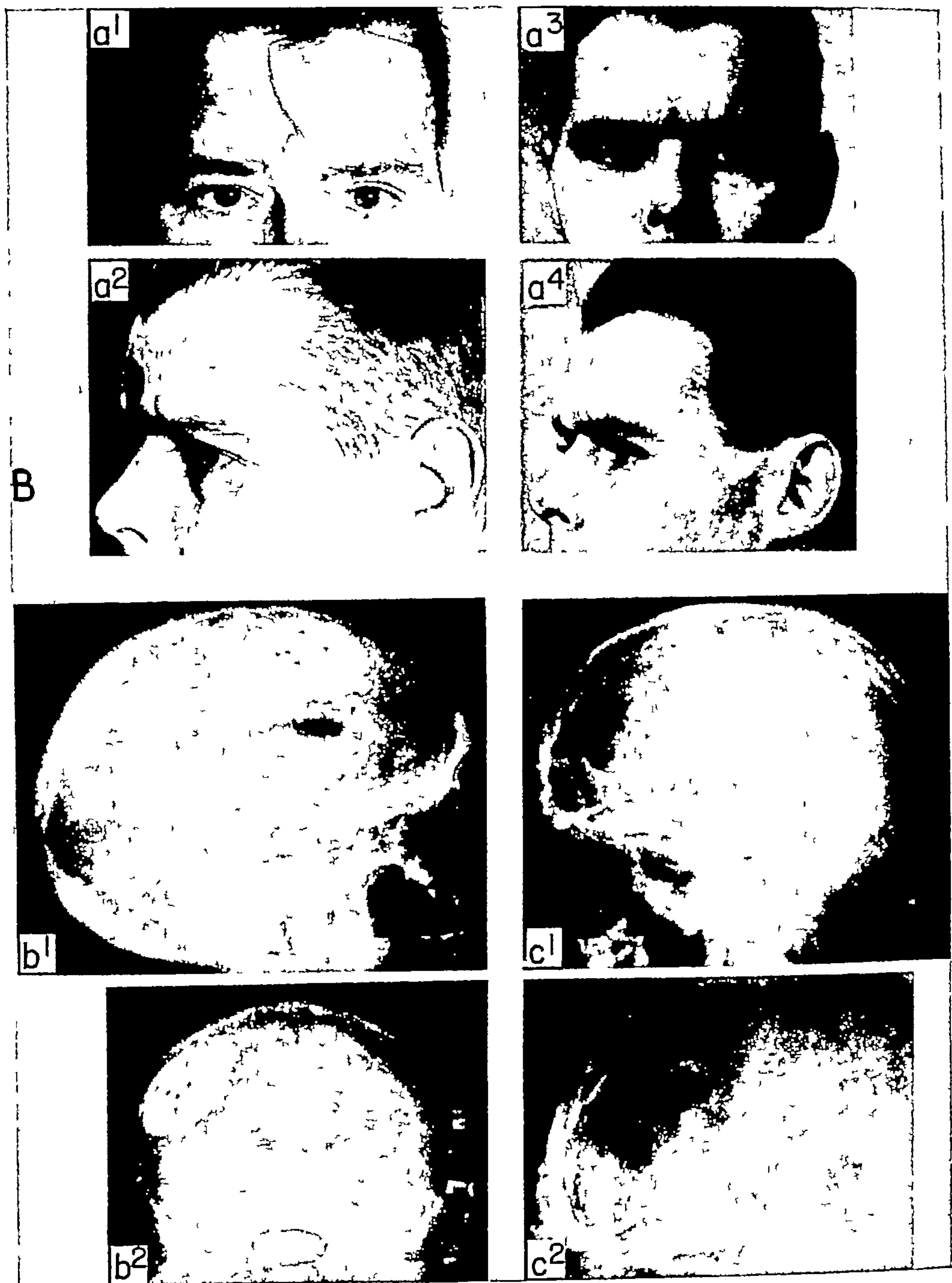


Fig 88 (*continued*) (*B*) Fracture and repair (*a*) with osteoperiosteal transplant in patient with cerebrospinal fluid rhinorrhea and escape of brain tissue from right nostril (*b*) Repair of frontal sinus defect with whole-thickness skull transplant obtained with trephine, trephined area repaired with tantalum (*c*) Brain abscess (visualized by Thorotrast), caused by patent cranionasal fistula, note communication with frontal sinus.

roentgenograms of the orbital and facial bones, is essential for a proper evaluation of the damage. The type of the incision is dictated by the character and severity of the fracture and the overlying laceration a coronal incision, a curved incision extending from the temple to the middle of the forehead, or an incision from eyebrow to eyebrow and another extending from the eyebrow on the involved side to the mid line and up the midline for 1 to 3 inches, a reversed *T* or *L* incision (Fig 90*A* *a* and *B* *a*) The incision should provide adequate exposure of the area. If the laceration is extensive, it can be extended by an appropriate incision and the depression explored through it. A low laceration can be extended as an incision of one or both eyebrows. As a rule, the incision should include the lacerated skin area, so that the borders of the tear can be excised completely

After exposing the area of depression (Fig 90*A* *b*), the comminuted pieces of bone are removed from the outer wall of the frontal sinus (Fig 90*A* *c*), the mucous membrane lining of the sinus is removed completely and its inner wall is carefully inspected If the posterior wall shows fracture lines or depression (Fig 88*A* *a*), the pieces of bone are removed and the frontal lobe dura is exposed Dural tears (Fig. 90*A* *d-e* and *B* *b-c*) should be exposed sufficiently to permit complete repair, this will prevent the development of a cranionasal fistula. The tears may be horizontal, vertical, or extend over the cribriform plate. A tear extending posteriorly must be followed to its end by careful dissection and inspection with a lighted retractor (Fig 90*B* *b*) All bleeding points are secured and coagulated, the dural edges are identified and carefully dissected off the base of the skull, and the edges of the tear are approximated with interrupted silk sutures. Usually, a dural tear over the cribriform plate can be repaired well by an extradural approach (Fig 90*B* *c*) After repair of extensive tears, by suture with interrupted silk sutures, or by a fascial transplant of fascia lata, temporal fascia, or subcutaneous fascia from the scalp, the area may be packed with iodoform gauze or gauze soaked in metaphen in oil. The wound is closed completely, 6 to 8 days later the wound

is reopened and the gauze pack removed In most instances, however, packing is not necessary

In some cases of severe depressed fracture of the frontal sinus neighborhood, the area is exposed by a coronal incision which permits the turning of a bone flap, the flap should cross and pass the midline for a distance of ¾ to 1½ inches (Fig 90*B, a*) If there is bleeding from the sagittal sinus it is controlled by applying a pad of Gelfoam, a pad of Cottonoid over it, and both held in place for several minutes, this combined with suction will produce hemostasis Some of the Gelfoam is left *in situ* at closure The tears in the dura are repaired (Fig. 90*B, a*), the dura is then opened, the dural flap being turned medially (Fig 90*B, b*) Veins crossing the cerebral surface to the sagittal sinus may have to be clipped or coagulated With a lighted retractor, the medial aspect of the hemisphere is retracted gently to expose possible midline dural tears more posteriorly

Cerebrospinal Fluid Rhinorrhea[2, 16, 30, 31, 49, 76]

The indication for surgery in acute cerebrospinal fluid rhinorrhea is usually the presence of a depressed fracture of the frontal sinus area, with or without laceration of the forehead The technics for open depressed fractures of this area have just been described In the absence of laceration or depression, or even fracture, in the frontal or the paranasal sinus regions, exploration of the anterior fossa may be indicated in patients with persisting cerebrospinal fluid rhinorrhea

The approach is by the usual coronal or frontal incision and an adequate bone flap This procedure is also indicated for recurrent rhi-

Fig 89 Management of depressed fractures of frontal sinus area (*A*) Fracture complicated by chronic patent cranionasal fistula (*a*) After 4 attacks of meningitis in patient with fistula and extensive paranasal injury, dural defect repaired by temporal fascia graft, no recurrence of meningitis (*b*) After 26 attacks of meningitis in patient with patent cranionasal fistula, dural defect repaired by approximating its edges with silk sutures, no recurrence of meningitis (*c*) Pneumocephalus due to patent cranionasal fistula which developed after dural defect was packed with Oxycel left *in situ*, fistula repaired by temporal fascia graft, skull defect later repaired with tantalum (*Continued on page 402*)

A





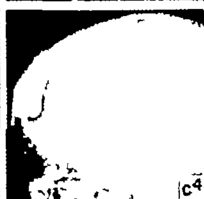












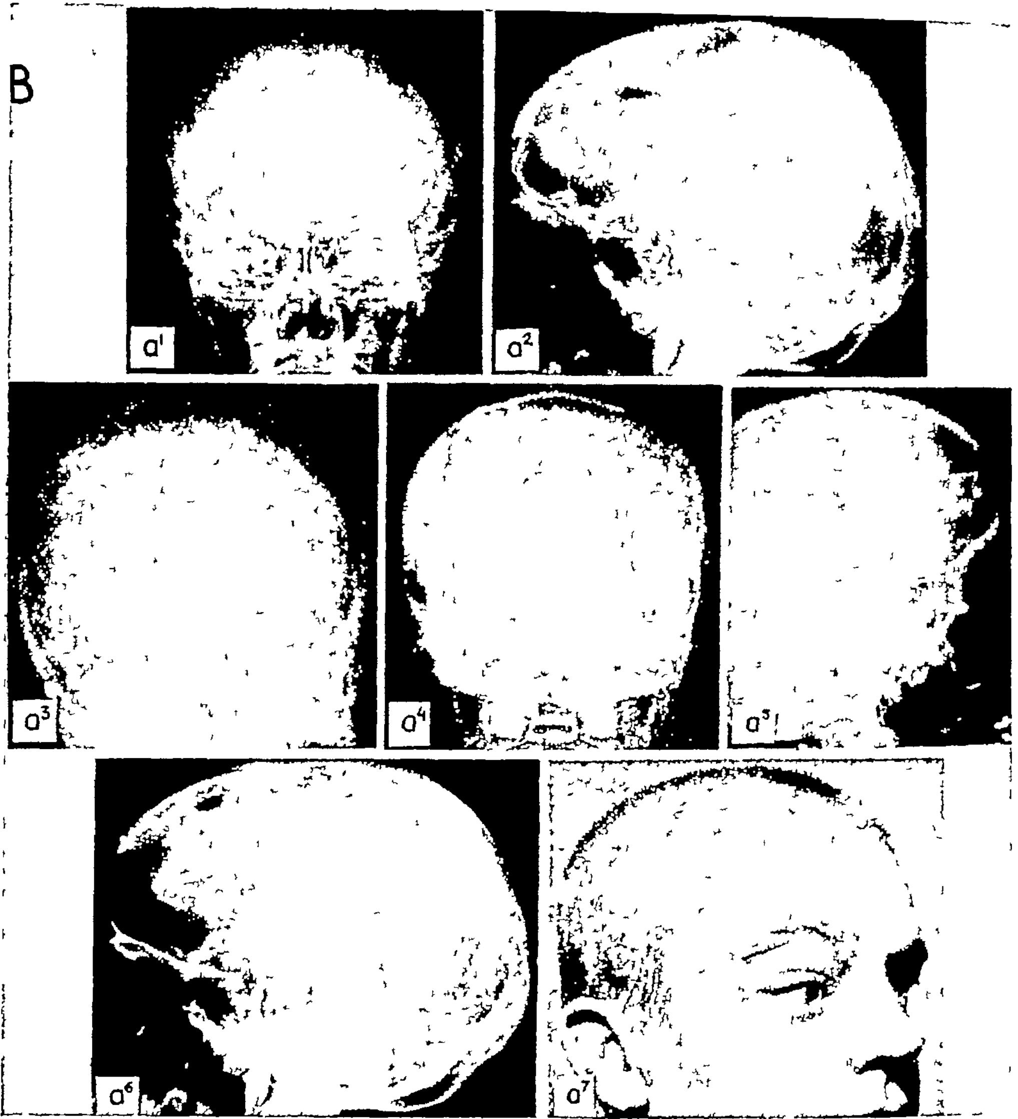


Fig 89 (*continued*) (*B*) Fracture of frontal sinus and orbital areas (a^1–a^2) Preoperative roentgenograms, patient desperately ill on hospital admission but conscious, severe pain in right forehead and bilateral papilledema, incomplete debridement at time of injury had resulted in chronic cerebrospinal fluid rhinorrhea, draining sinus in right eyebrow, and pneumocephalus (a^3) Angiogram showing brain abscess, all suppuration removed through trephine openings superiorly and posteriorly, after debridement and removal of necrotic dural and cerebral tissue (a^4–a^6), penicillin instilled into abscess cavity twice.

norrhea, in such cases, complicating infections (e g meningitis or brain abscess) which may be present must be controlled before exploration is undertaken

Recurrent rhinorrhea is a clear indication of the presence of a cranionasal fistula, and the surgical procedure to be used for its elimination is dictated by the size and location of the fistulous opening As a rule, cerebral herniation into the bone defect has occurred through a dural tear The more medial and posterior the site of the fistula, the more difficult it is to reach and close it.

We have found the following procedure to be effective (*1*) The fistula is adequately exposed by means of a bifrontal bone flap, particularly if the cranionasal fistula is at the midline. (*2*) The fistula should be exposed both extra and intradurally by thorough dissection of the area and removal of the cerebral herniation, which has become sealed off around and into the dural defect, reflection of the dura from the crista galli provides more adequate exposure in some cases. (*3*) The site is inspected for bone fragments, and they are removed (*4*) The dural opening is closed by a graft of temporal or pericranial fascia or fascia lata sutured to the dura Other methods of closing the fistula, suggested or used, are (*a*) by a reflected portion of the falx cerebri (*b*) by an osteoperiosteal transplant from the tibia, (*c*) by cauterizing the fistula and plugging it with a muscle transplant, (*d*) by a fascia lata transplant placed intradurally without suture In our experience, suture of a fascial transplant is the safest method of repairing the dural defect, in few cases has it been impossible to suture the graft to the reflected borders of the dural opening Should we find this condition, we would be inclined to attempt suture of the graft to the free edge of the lesser wing of the sphenoid and to the reflected border of the dura more anteriorly and medially until a good snug closure is obtained (*5*) Hemostatic materials (Gelfoam, Oxycel) are not reliable substitutes for a snug closure by a fascial graft, but a layer of such material may be placed between the dura and the base of the skull to good advantage after the defect has been closed. (*6*) After dissection the bone defect

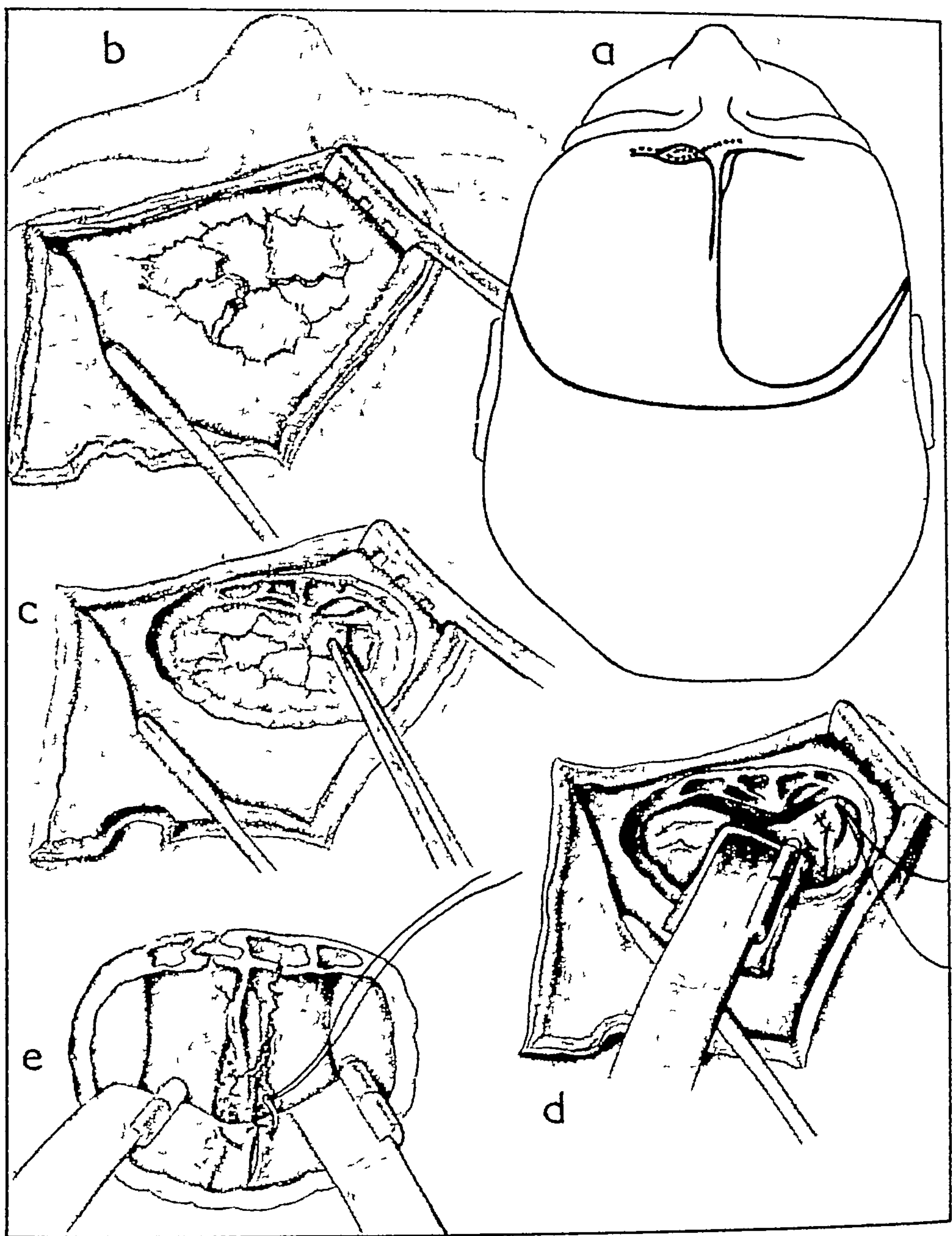


Fig 90 Management of open depressed fracture of frontal sinus area (*A*) Types of incision (*a*) (*b*) Exposure of depressed area (*c*) Removal of bone fragments (*d–e*) Exposure and repair of dural tears.

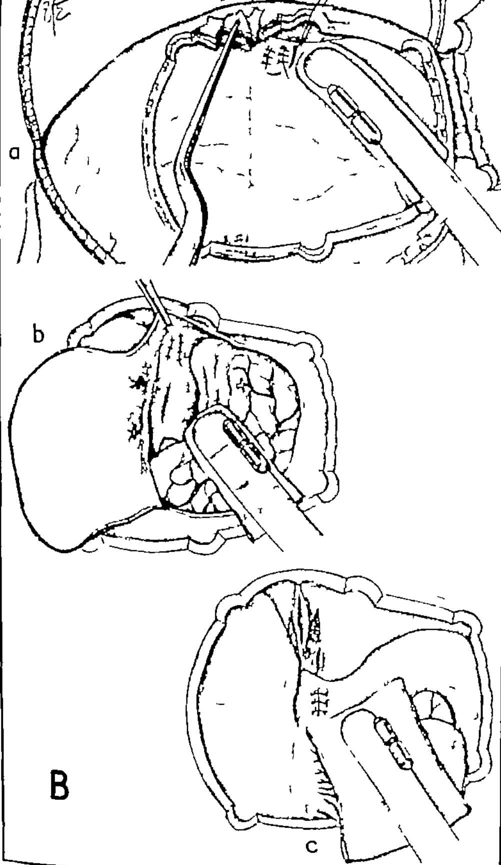


Fig 90. (*continued*) (*B*) Coronal incision and bone flap, exposing dural tears (sutured) (*a*) (*b*) Dural flap turned medially exposing dural tear over cribriform plate; lighted retractor over medial aspect of hemisphere. (*c*) Repair of posterior dural tear

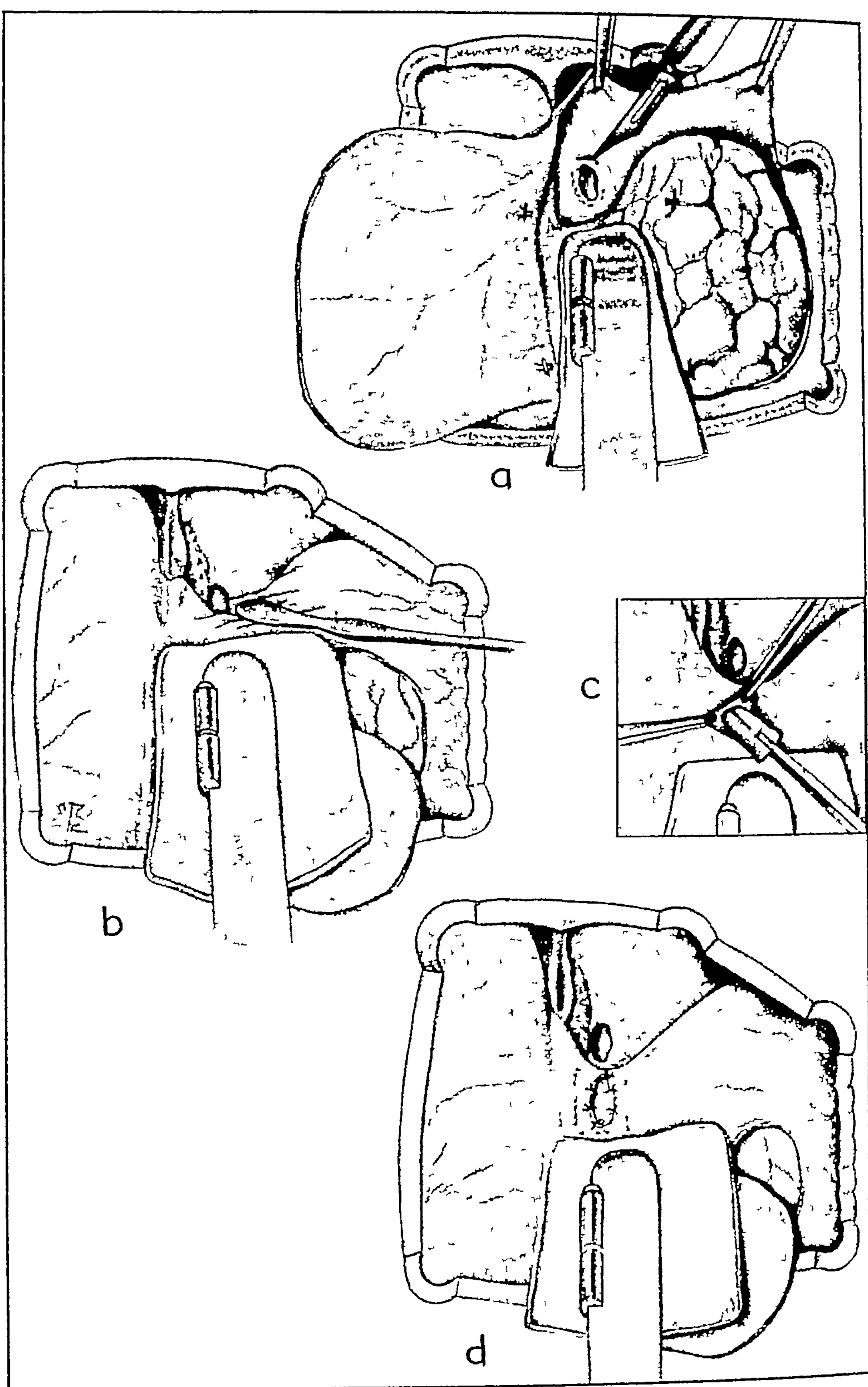


Fig 91. Management of patent cranionasal fistula (*a*) Bone flap crossing midline, dura opened, veins from cerebral surface to sagittal sinus clipped and cut, brain retracted, anterior fossa exposed (*b–c*) Dura replaced over brain and area of defect exposed extradurally, dural defect freed from defect in skull base, with care taken not to tear dura further (*d*) Repair of defect with temporal fascia graft introduced into interior of dural sac and sutured into place.

may be sealed with methyl methacrylate to insure a successful repair of the fistula. The plastic, soft and pliable before it sets, may be used to plug the hole, it solidifies as it sets and a snug closure is obtained We have used this method in 2 cases with excellent results. (7) If a good repair cannot be accomplished at the first attempt, complete closure of the fistula should be tried once more at a later date.

Penetrating Injuries

The treatment of all penetrating injuries includes early, meticulous debridement and excision of all devitalized or contaminated tissue in order to prevent infection, treatment of a complicating hematoma, and repair of the skull defect(s) The extent of debridement, and the procedures for subsequent management and repair, are governed by the size, type, and velocity of the injuring object and the area injured With penetrating injuries, one must be ready at any moment to revise treat ment should the patient s condition change for the worse. After the initial treatment, such changes occur mainly from intracranial hem orrhage or infection Pre and postoperative roentgenograms are man datory in every case of penetrating injury Antibiotics (1,000,000 to 2,000,000 units penicillin) and sulfonamides (8 Gm sulfathiazole or 4 Gm. Gantrisin) per day are administered routinely in the absence of frank infection. Larger dosages must be used if meningitis or brain abscess is present, and the drug sensitivity of the organism should be established Occasionally, a contaminating organism proves resistant to routine antibiotic therapy, the organism must then be cultured in order to identify it and thus establish its sensitivity to other therapeutic agents. Since World War II, in which the gram negative organisms, including the Psuedomonas, caused such serious complications, newer antibiotics (Neomycin, streptomycin) parenterally have been found effective in such infections.

Ideally, all wounds should be debrided within 24 hours, but when facilities for such surgery are not available the debridement may be

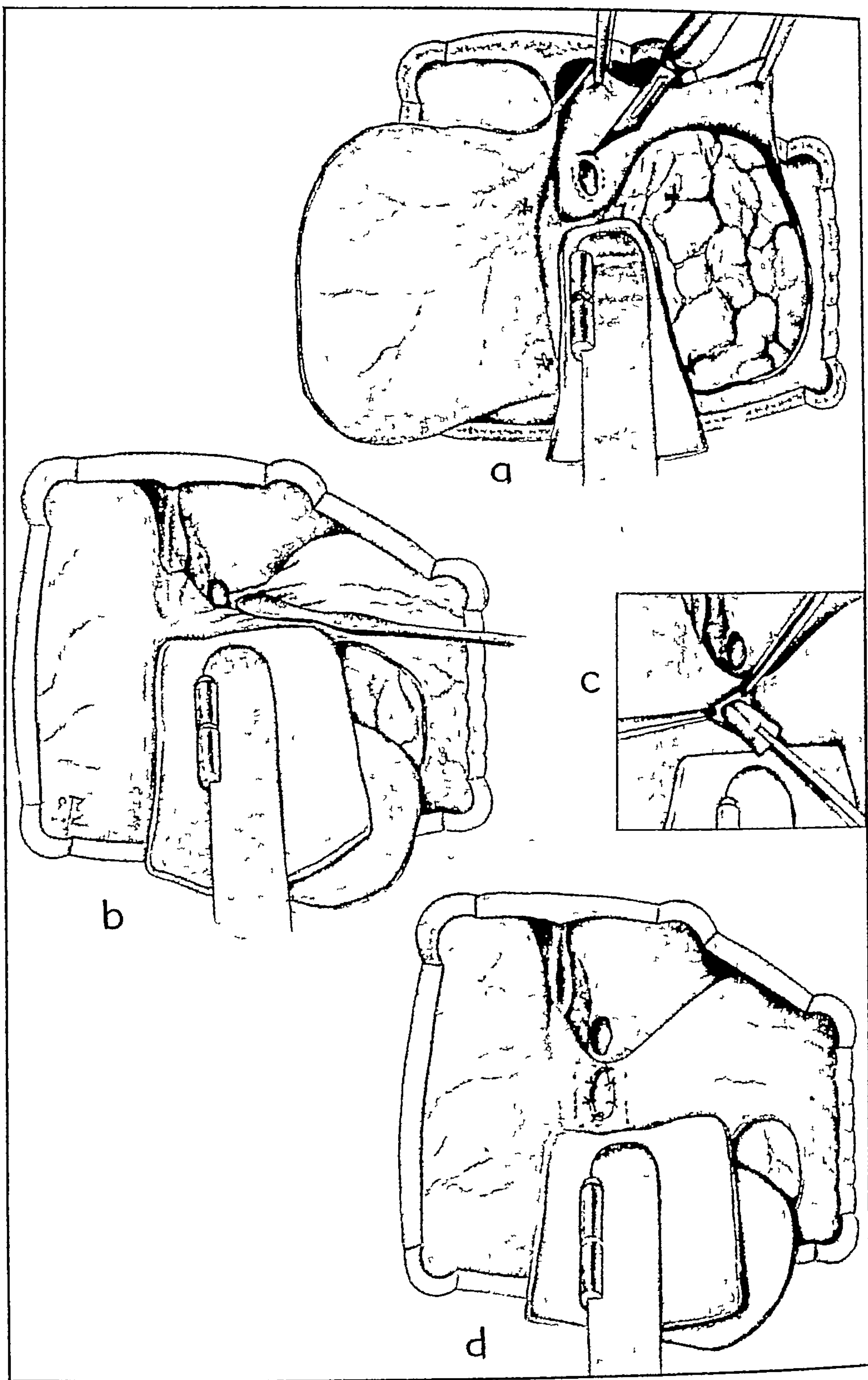


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may be sealed with methyl methacrylate to insure a successful repair of the fistula. The plastic, soft and pliable before it sets, may be used to plug the hole, it solidifies as it sets and a snug closure is obtained We have used this method in 2 cases with excellent results. (7) If a good repair cannot be accomplished at the first attempt, complete closure of the fistula should be tried once more at a later date.

Penetrating Injuries

The treatment of all penetrating injuries includes early, meticulous debridement and excision of all devitalized or contaminated tissue in order to prevent infection treatment of a complicating hematoma and repair of the skull defect(s) The extent of debridement, and the procedures for subsequent management and repair, are governed by the size, type, and velocity of the injuring object and the area injured With penetrating injuries, one must be ready at any moment to revise treatment should the patient s condition change for the worse. After the initial treatment, such changes occur mainly from intracranial hemorrhage or infection. Pre and postoperative roentgenograms are mandatory in every case of penetrating injury Antibiotics (1,000,000 to 2,000,000 units penicillin) and sulfonamides (8 Gm. sulfathiazole or 4 Gm. Gantrisin) per day are administered routinely in the absence of frank infection. Larger dosages must be used if meningitis or brain abscess is present, and the drug sensitivity of the organism should be established Occasionally, a contaminating organism proves resistant to routine antibiotic therapy, the organism must then be cultured in order to identify it and thus establish its sensitivity to other therapeutic agents. Since World War II, in which the gram negative organisms, including the Psuedomonas, caused such serious complications, newer antibiotics (Neomycin, streptomycin) parenterally have been found effective in such infections.

Ideally all wounds should be debrided within 24 hours, but when facilities for such surgery are not available the debridement may be

delayed In most cases, delayed primary debridement is attended by primary healing, particularly if antibiotic and sulfonamide therapy has been instituted When wounds are frankly infected, however, a secondary debridement is necessary, since in such cases bone fragments and foreign matter, accompanied by cerebritis or brain abscess, are usually present

By Low-velocity Objects

These are the injuries commonly seen in civilian life The object may be a knife, an ice pick, any sort of sharp tool or object, or a low-velocity missile, such as a bullet from an air gun or a 22 short rifle Essentially, the management of all these injuries is the same debridement, prevention or cure of infection, management of complicating intracranial hemorrhage, and repair

If there are no bone fragments in the brain, a patient with a low-velocity penetrating injury is observed to establish the possible presence of intracranial infection or hemorrhage The site of the laceration provides a clue to the possibility of a tear in a major blood vessel, if there is such a possibility, exploration should be undertaken before there are signs of cerebral involvement If the foreign body is still in the cranial cavity, it should be removed if at all possible The continued presence of the object may cause infection and/or abscess formation

Among the more common low-velocity penetrating injuries are stiletto and knife wounds The end of the knife is frequently broken off, and if skull roentgenograms are not made the metal fragment may be completely overlooked (*see* Fig 66*A*) The edges of the scalp laceration are excised and the skull is exposed, the edge of the metal may then be seen If the metal is easily dislodged, it is removed, if not, a button of bone surrounding the metal is sawed through and the foreign body is excised The button of bone may be replaced before the scalp wound is closed If there is undue bleeding after removal of a foreign

body, a more extensive craniotomy may be necessary to secure the bleeding points.

A rare type of low velocity injury is penetration by an umbrella end or a long stick through the nostril and into the base of the brain After removing such a foreign body, exploration may be indicated for massive intracranial bleeding or persistent cerebrospinal fluid rhinorrhea.

The possibility of massive intracranial hemorrhage with low velocity penetrating wounds should always be kept in mind (*see* Fig 50*a*) After indicated diagnostic procedures, management by an adequate craniotomy may be successful in many instances

By High-velocity Missiles (Figs. 92-94)

With the modifications imposed by the type and site of the wound, the technic is as follows

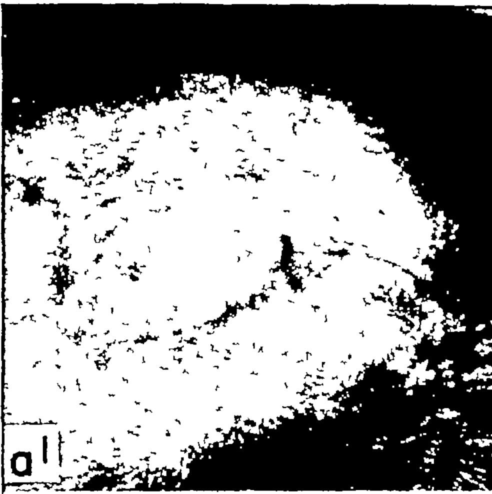
(*1*) A long, curved incision is made so as to excise the entire lacerated area or areas of scalp (Fig 94) In the case of a frontal injury, the coronal incision should be extended into the temporal area anteriorly to permit approximation of the skin in the midforehead after excision of the lacerated scalp (Fig 94*A* *a*)

(*2*) The skull is exposed at the wound of entry and at the one of exit, if there is one (Fig 94*A* *c*)

(*3*) If the depressed fracture has not penetrated the dura, the bone fragments are removed, and the dura is opened if it is blue because of underlying hemorrhage. If a liquid clot or subdural accumulation of cerebrospinal fluid is found, drainage is accomplished through a wider exposure (Fig 94*A* *b*)

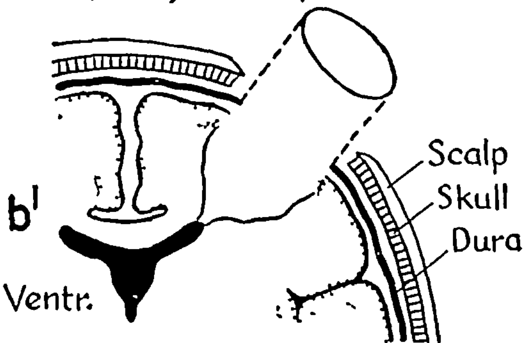
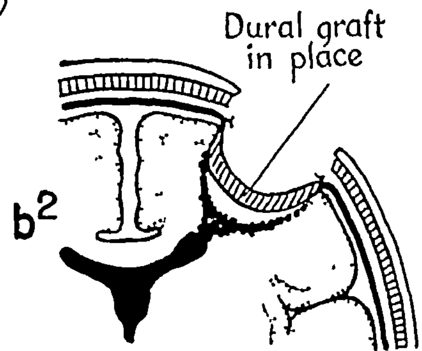
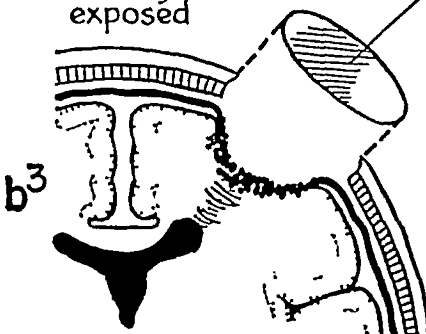
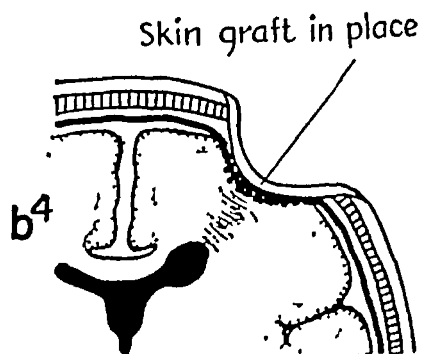
(*4*) If the dura is lacerated, sufficient bone must be removed to expose healthy looking dura throughout the craniectomy, with the dural tear in the middle (Fig 94*A* *c*), care must be taken during the exposure not to catch dura and bone in the mouth of the rongeur and thus destroy undamaged dura

(*5*) The lacerated edges of the dural tear are debrided, and the under


a1

a2

a3

a4

Temporary dural graft (fascia lata)
Scalp
Skull
Dura
b1
Ventr.

Dural graft in place
b2

Dural graft removed
Granulating surface exposed
Split-thickness skin graft
b3

Skin graft in place
b4

lying brain is inspected, necrotic cerebral tissue may be extruding from the missile tract as well as fragments of clotted blood

(6) The missile tract is identified, cleansed completely by irrigation with saline, and cautiously inspected with an exploring finger, this can be done gently without damaging the tissues Injected saline may become trapped in the tract or in the intracranial space, careful manipulation of the tissues permits the escape of the entrapped fluid

(7) Devitalized cerebral tissue is excised by suction at 30 pounds per square inch, with caution not to excise normal tissue, particularly in the speech or motor areas, devitalized tissue comes out easily, whereas normal tissue is resistant, and the difference between the two can be distinguished with the suction tip by an experienced surgeon.

(8) If a metal fragment is easily accessible, it should be removed, if its location would necessitate another craniectomy, or its removal might cause additional cerebral damage, it may be wiser to leave it alone until indications arise for its removal In any case in which a bullet or shell fragment is at a distance from the wound of entry, another craniectomy is necessary

(9) The wound is inspected a final time and tested for hemostasis by compression of the jugular veins of the neck. All bleeding points must be carefully coagulated, Gelfoam or Oxycel may be used, but in most cases it should be removed as soon as hemostasis is complete.

(10) The dura is closed tightly with the help of a transplant of temporal or pericranial fascia, fascia lata, or preserved fascia, and silk sutures.

(11) The scalp is closed over the skull defect it may be necessary to use a flap of scalp, in which case a skin graft is used to cover the area from which the flap has been obtained

(12) The skull defect is repaired after the wound has healed completely and 6 to 12 months have passed without complicating infection

Fig. 92. Management of severe penetrating head injury (*a*) Before and after tantalum cranioplasty (*b*) Temporary fascial graft for dural defect after complete debridement (b^1–b^2) and split-thickness skin graft 10 days later (b^1–b^1)

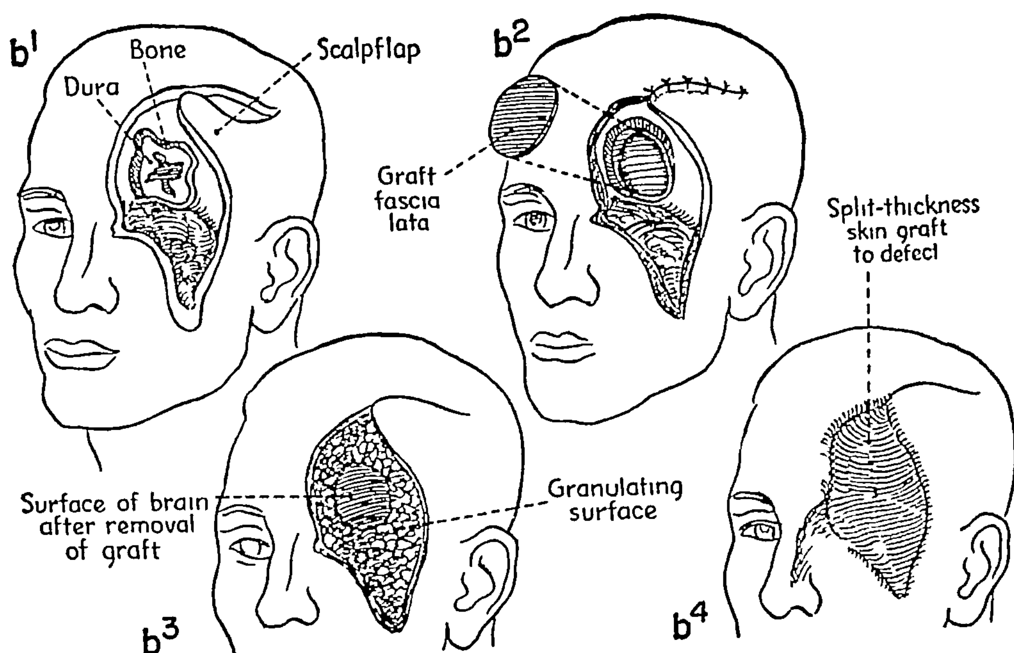


Fig 93 Management of severe orbitocranial injury (*a*) Orbital contents completely destroyed and frontal bone badly shattered (a^1) Infection 10 days after inadequate debridement (a^2) Ten days after secondary debridement and temporary repair of dural defect (a^3–a^4) Skin graft for permanent repair. (*b*) Diagrams of repair. (b^1) Dural tear found on secondary debridement (b^2) Dural defect closed with temporary fascial graft. (b^3) Cerebral surface after removal of temporary graft (b^4) Split-thickness skin graft.

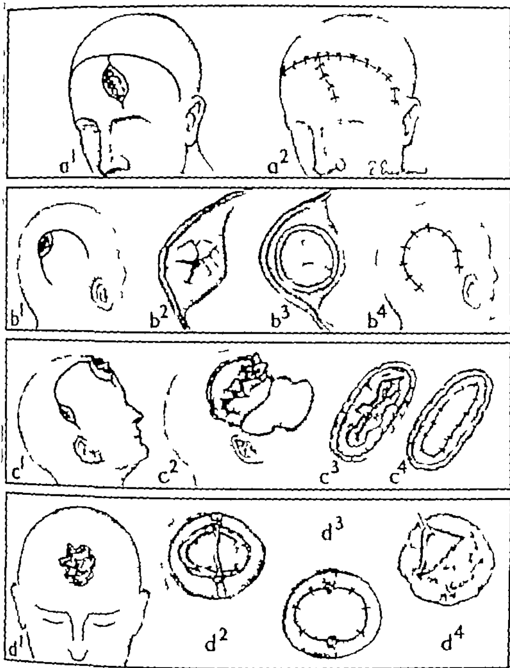


Fig. 94 Management of penetrating head injury (*A*) Frontal injury (*a*) coronal incision and suture of scalp. (*b*) Skull penetration without dural penetration incision (b^1) exposure of depressed fracture (b^2) removal of bone fragments (b^3) and suture of scalp (b^4) (*c*) Tangential injury extensive fragmentation of bone, dural tear and destruction of cerebral surface tissue incision (c^1) exposure of fracture (c^2) exposure of dural tear and cerebral destruction (c^3) and repair of dural defect by fascial graft (c^4) (*d*) Frontal injury destruction of scalp, bone, and dura (d^1) double ligation of sagittal sinus after complete debridement (d^2) temporary fascial graft for dural defect (d^3) and removal of temporary graft in preparation for skin graft (d^4) (*Continued on next page*)

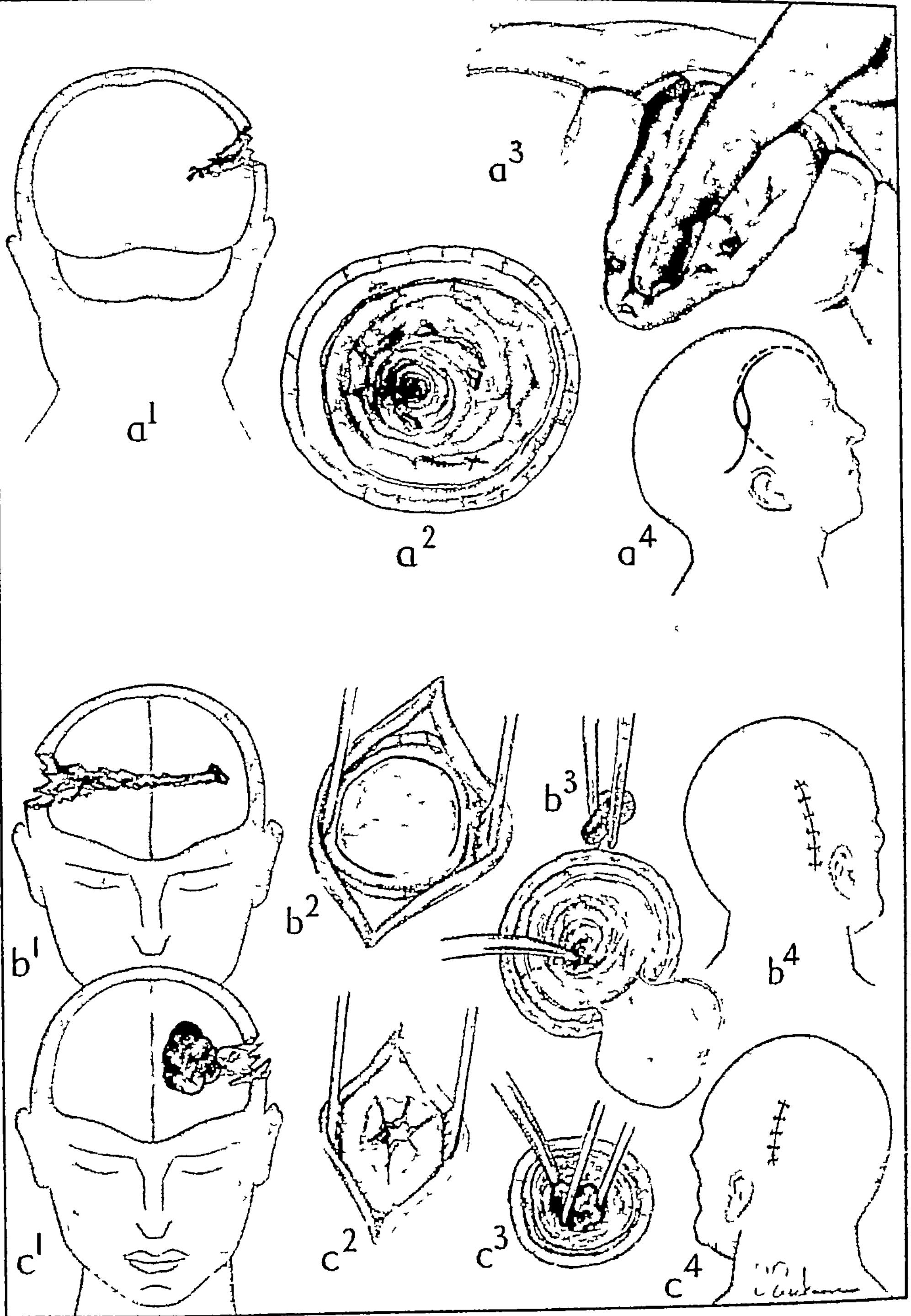


Fig 94 (*continued*) (*B*) Penetrating injury (*a*) with shattering of bone along missile tract, note exploring finger in tract (a^3) and lines of incision (a^4). (*b*) Bullet injury, tract of bullet, lodged near pericranium on opposite side (b^1), exposure (b^2), removal of bullet (b^3), and closure of wound (b^4) (*c*) Management of hematoma in missile tract

Orbitocranial Injury (Fig 93)

In such injuries, the skull and the orbital area are debrided at the same time, and most satisfactorily by a team including an ophthalmologist and a neurosurgeon

The orbital rim should be preserved so far as possible, and the medial superior portion of the orbit retained in order to keep the pulley of the superior oblique muscle intact. The roof of the orbit and the cribriform plate area must be carefully inspected for dural tears, which may cause a craniomasal fistula

Appropriate scalp incisions are used to expose the dura. Free bone fragments are removed If the dura is extensively damaged, and there is loss of scalp and orbit, the method of choice is to separate the dural sac completely from the surrounding tissues. The dural wound and the underlying cerebral tissue are debrided and a temporary watertight fascial graft is applied, this may be a fascia lata graft. After 10 or more days, when the cerebral surface is covered with granulation tissue, the graft is removed, and the defect over the brain is covered with a split thickness skin graft At a later date, a more appropriate repair is obtained by reconstructive surgery

Dislocation of the globe, which may result from injury of the orbital floor, is managed in some cases by elevating the eyeball until it is in alignment with the other eyeball.

Uncal Herniation (Fig 95)

Cushing's[14] well known subtemporal decompression technic has continued to be a useful procedure for the past 50 years but as originally described it is of limited usefulness because the opening in the temporal area is small, due to the type of incision used and the presence of the temporal muscle. The original subtemporal decompression has been modified so that it can be used in an osteoplastic craniotomy the flap remains hinged to the temporal muscle and adequate amounts of bone

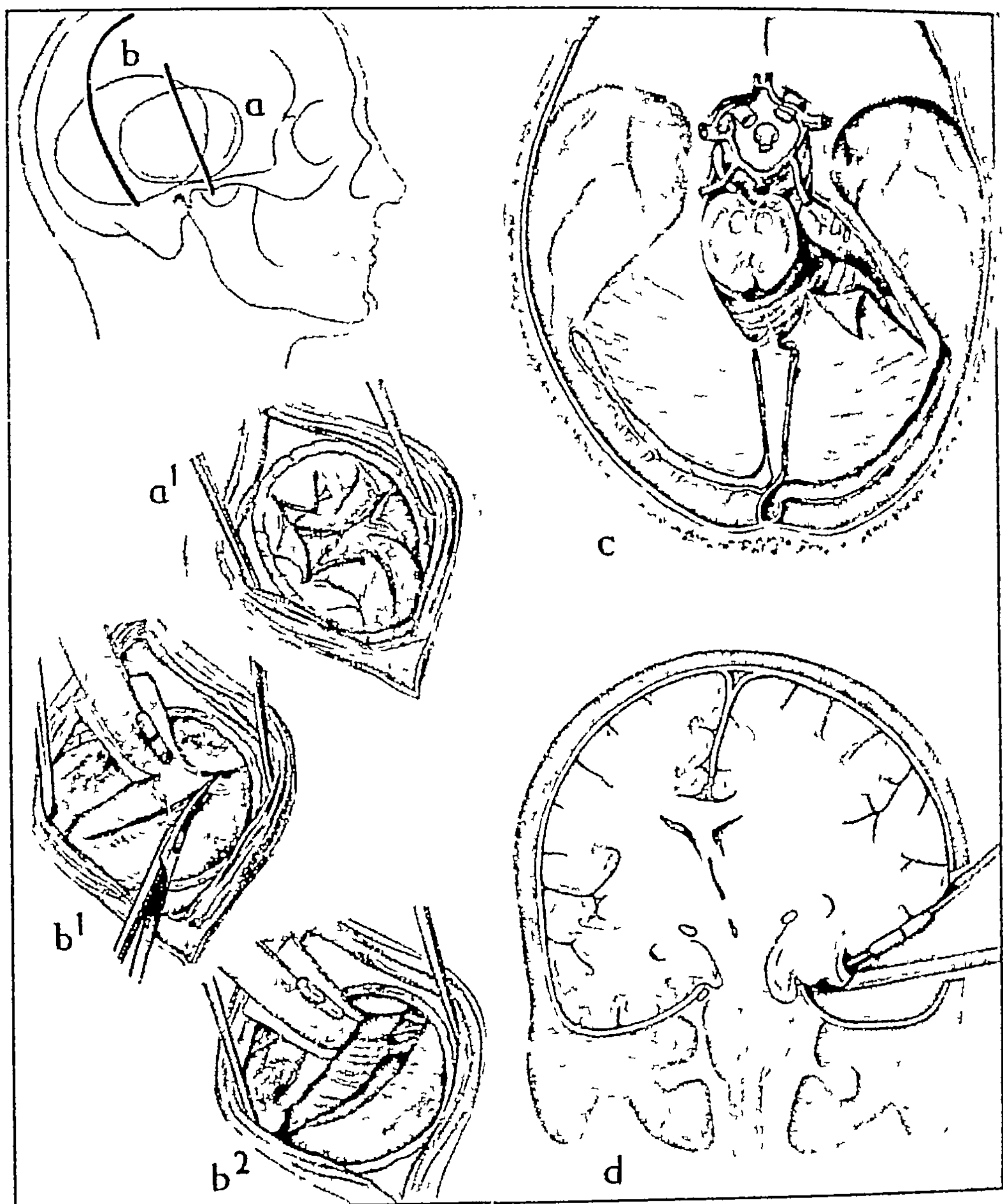


Fig 95 Decompression procedures (a, a^1) Cushing's subtemporal decompression (b, b^1–b^2) Tentorial decompression, incision of scalp and tentorium just posterior to superior border of petrous bone (c) Structures at base, intact tentorium on left, incised tentorium on right, oculomotor nerve passing under posterior cerebral artery, posterior cerebral, posterior communicating, and basilar arteries visible, note left side of brain stem pushed against incisural border (d) Typical uncal herniation on right.

are removed from the flap and the part of the temporal bone which forms the lateral boundary of the middle fossa.

The valuable studies of Vincent and associates[63] introduced an operative procedure for uncal herniation consisting of excision of a portion of the temporal lobe including the uncus. Munro and Sisson,[47] too suggest the value of tentorial decompression in cases of uncal herniation. There is general agreement on the importance of the speediest possible removal of the cause of the herniation, particularly if the diagnosis is established early. A valuable prophylactic measure for preventing herniation is the removal of an extradural, subdural, or intracerebral hematoma and a tentorial decompression if necessary. Prompt surgical treatment may be lifesaving. In some cases, evacuation of the hematoma and removal of the uncus may suffice; in others, in which the herniation of the temporal lobe or the brain stem has been present long enough to produce irreversible changes, excision of the uncus and medial portion of the temporal lobe may be worth while.

The lower portion of the temporal lobe, including the uncus, may be excised by using either of two incisions (Fig. 95*a*-*b*). The tentorium is incised just posterior to the superior border of the petrous bone, the temporal and occipital portions of the hemisphere are gently elevated with a lighted retractor over a piece of Cottonoid, the tentorial sheath is carefully visualized, and the falx cerebelli is incised (Fig. 95*b*'). When the incision is completed down to the incisural border, cranial nerves V, III, and possibly VI, may be seen in the more medial portion of the incision. The uncus may be removed as the tentorium is being incised. Complete hemostasis is essential since large venous channels may be encountered over the inferior surface of the temporal lobe and over the occipital lobe. The compression of the brain stem toward the left (Fig. 95*c*) is the type of herniation producing the paradoxic paralysis on the same side as the hematoma.

The inferior portion of the temporal lobe can be excised by suction, and the area of the incisural border then may be carefully inspected. In some cases, it may be possible to elevate the temporal lobe without ex

cising any part of it, it may also be possible to elevate the uncus which may be herniating into the posterior fossa This is more apt to be the case when the hematoma has been removed and the increased intracranial pressure has in large measure been relieved

Repair of Skull Defects[26, 28, 74, 88] *(Figs. 96-98)*

Attempts to repair defects in the skull go back to the beginning of our century The materials that have been tried are legion silver, aluminum, vulcanite,[19] bone from various animals,[3] boiled cadaver bone,[28, 61] homologous bone from various parts of man's anatomy, including the skull, rib, tibia, scapula, and ilium,[8, 39, 51, 67, 75] inanimate substances such as stainless steel,[77] tantalum,[21, 37, 47, 69, 81, 84, 87] celluloid,[58] Vitallium,[6 22, 65] and methyl methacrylate[18 70 78] The use of cadaver bone and rib and other bone transplants was abandoned when it was found that they were absorbed within a comparatively short time — 18 months or less (Fig 96) Frozen bone flaps from human bone bank have also been advocated[1, 59 73] Only three materials are now in common use for the repair of skull defects, (*1*) the homologous osteoperiosteal transplant from the patient's skull, which usually remains viable and is the material of choice in certain cases, but is now infrequently used, (*2*) tantalum, also less frequently used since the introduction of methyl methacrylate, and (*3*) methyl methacrylate (Plexiglas, Lucite, Perspex), a plastics material

During a 10 year period (1945-1955), 105 cranioplasties were performed, 91 were for head injury, 11 for tumors of brain and skull, 2 for skull defects with intracerebral hemorrhage, and 1 for a defect after extradural abscess Osteoperiosteal transplant repair was done in 4, the transplant having been obtained from the patient's skull; 80 were repaired with tantalum, and 21 were repaired with methyl methacrylate

In 3 cases, a dented tantalum plate was removed, reshaped and replaced, infection in 5 cases necessitated removal of the plate (in one

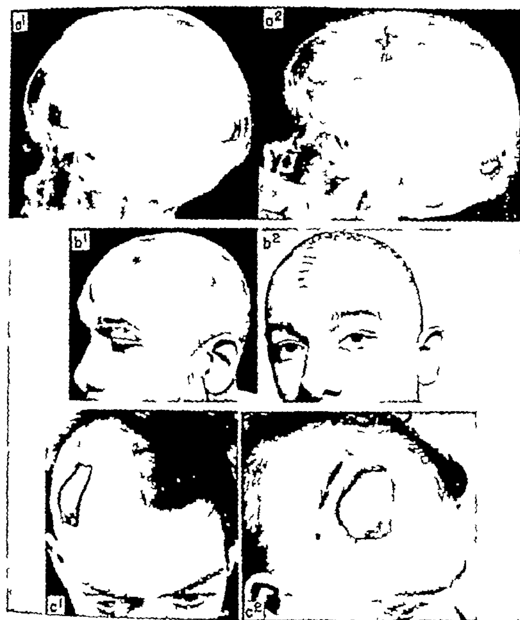


Fig. 96 Complications of cranioplasty (*a*) Absorption of bone after repair with boiled cadaver bone. (*b*) Tantalum plate deformed by blow plate removed and replaced after correction of its contour (*c*) Unsuccessful tantalum cranioplasty, plate should have been removed long before this stage was reached.

of these, methyl methacrylate cranioplasty was successfully used later, a second insertion of a tantalum plate was well tolerated in 2 others while in the remaining 2, nothing more was done because the cosmetic appearance was satisfactory) In another case, in which cranio-

plasty was done elsewhere, the appearance of the patient was better after the plate was removed

In 21 patients, methyl methacrylate repair was used, 9 of them had extensive repairs of forehead defects Thus far, no difficulties have been encountered and there has been no need to remove the plate

Osteoperiosteal Transplant[32, 42, 62] (Fig. 97)

This method is particularly indicated for small defects over the paranasal or frontal sinuses A full-thickness button of bone is removed with a 1 or 2 inch trephine from the skull (Fig 97*a*) The pericranium is puckered, to save its attachment to the outer surface of the skull (Fig 97*b*), the central pin of the trephine should engage the skull in the middle of the fascial lining so as not to tear the pericranial attachment After the button of bone is removed it is cut down to the proper size with a rongeur (Fig 97*c*) The area of defect is shaped and prepared to receive the transplant, the button of bone is placed in position, and the pericranium of the transplant is sutured to the pericranium at the border of the defect (Fig 97*d*) The skull defect caused by the removal of the transplant is repaired with a tantalum plate (Fig 98)

The possibility of a good "take" and the unlikelihood of infection make the osteoperiosteal cranioplasty a somewhat preferable method to that with an inanimate material, such as tantalum or methyl methacrylate But because methyl methacrylate permits complete obliteration of the frontal sinuses, and the likelihood of infection is much smaller than with tantalum, the plastics material is used most frequently at present

Complete hemostasis is essential Small quantities of antibiotics are used locally (1,000,000 units of penicillin in 10 cc solution) to minimize the possibility of infection Fluid may collect under the scalp after complete wound closure After 3 or 4 days, the entire collection can be removed, under strict asepsis Occasionally, it may be necessary to repeat the maneuver once or twice, and, rarely, three times

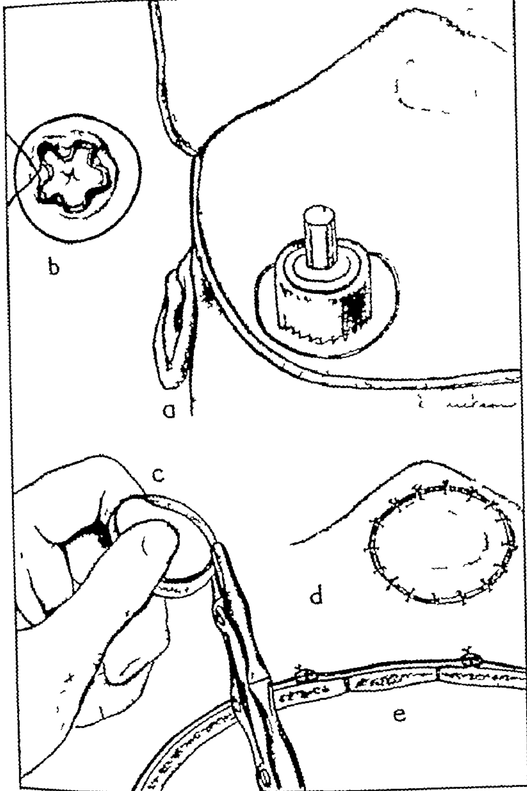


Fig 97 Cranioplasty with osteoperiosteal transplant. (*a*) Area of defect near base of nose exposed by scalp flap full-thickness bone being removed from outer skull table by circular saw with central pin (modiolus) engaging skull in middle of fascial lining. (*b*) Pericranium puckered to save its attachment to outer skull surface. (*c*) Transplant cut down to size by rongeur (*d*) Transplant in place in skull defect, with its pericranium sutured to pericranium of edge of defect. (*e*) Diagram of repair and attachment of pericranium.

Tantalum Cranioplasty (Figs. 98-99)

The methods used at first have been considerably simplified in recent years While the earlier method, which included making a cast of the defect, templates to fill the defect until both sides of the skull were even, and a tantalum plate to cover the area, is still used at times for a large defect, the following, simpler technic has been found effective for all but the very large defects

(*1*) A paper pattern is made of the defect as it can be felt through the scalp

(*2*) The contour of the homologous area on the opposite side of the skull is noted, so that the plate can be correctly curved

(*3*) The paper pattern is laid on the tantalum and the border is outlined, the frontal, medial, and lateral borders of the plate being marked to facilitate orientation later The plate should be just large enough to allow about ¼ inch overlap on the bone around the defect; this gives a better fit, minimizes warping of the edges, and facilitates shaping, particularly of such curved areas as the forehead and temporal region.

(*4*) The plate is placed on a piece of lead and hammered out to the desired contour with a ball-peen hammer, here much depends on one's ability to visualize the contour and reproduce it with the hammer.

(*5*) Holes of ⅛ inch diameter are punched out around the periphery and throughout the plate at regular intervals, the sharp edges of the holes are smoothed and flattened by gentle hammering on an anvil, and the border of the plate is filed and ground smooth. In the correctly fashioned plate, the entire border is slightly but sharply turned inward; the snug fit which this insures prevents later loss of the plate as a result of pressure necrosis of the scalp

(*6*) The plate is washed with ether and with alcohol and autoclaved, and is then ready for use

With this technic, little shaping need be done in the operating room Gentle hammering will accomplish the slight adjustments that are occasionally needed for a snug fit If the plate is too large for the de-

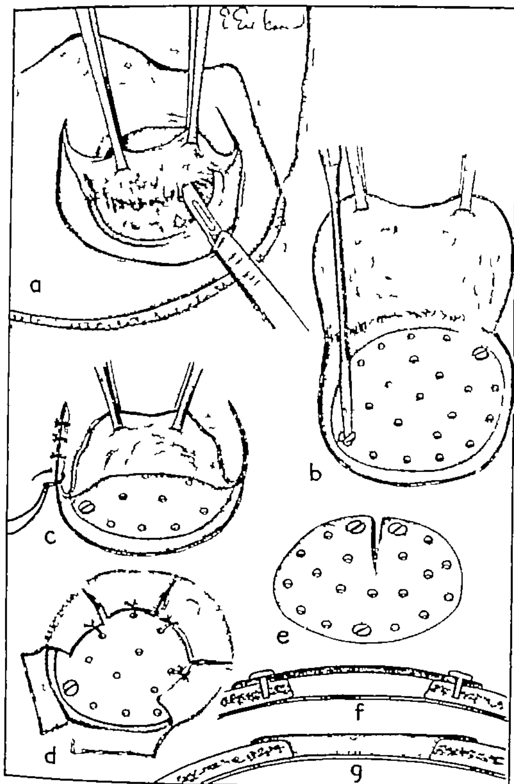


Fig. 98. Tantalum cranioplasty (*a*) Exposure of defect and separation of pericranium from dura. (*b*) Tantalum plate being screwed into place. (*c–d*) Different methods of attaching pericranial layer to tantalum plate. (*e*) Wedge cut out of plate to secure better fit. (*f–g*) Cross section of repair showing initial dead space between plate and dura (*f*) and absence of dead space with methyl methacrylate repair (*g*)

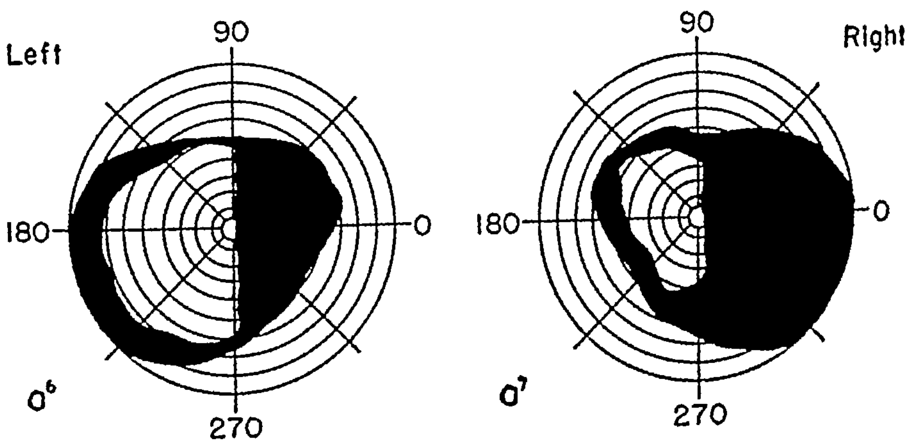


Fig 99. Old open depressed skull fracture of left occipitoparietal area, skull defect repaired by tantalum plate (a^5), note communicating porencephalus (a^1–a^4) and right homonymous hemianopsia (a^6–a^7).

fect, a triangular wedge (or wedges) is cut from the edge of the plate and the borders are brought down and secured to the skull (Fig 98e)

To repair the skull defect (1) The area of defect is exposed and the pericranium is separated from the dura (Fig 98a), this separation may be impossible in some cases (2) The tantalum plate is placed over the defect, with the border of the plate inserted under the pericranium and secured to the skull with wire or tantalum screws (Fig 98b) (3) The layer of periosteum is pulled over the plate and sutured with interrupted silk sutures (Fig 98c) Another method of dealing with the pericranium over the defect is to leave the pericranium in place but incise it around the edge of the defect and reflect it back after cutting into it at several points, and suture it to the plate which has been screwed in (Fig 98d), it is important that the entire border of the plate be covered with pericranium. In certain areas, e g the temporal temporal muscle or fascia is used for coverage.

In the postoperative period, usually as a result of incomplete hemostasis during the operation, some fluid may collect under the scalp and over the plate If the fluid has not been resorbed within 3 to 4 days, it should be removed by aspiration under strict asepsis.

The presence of infection under the scalp is a contraindication to tantalum cranioplasty for about a year after complete healing of the wound. As a rule, the plate is not tolerated if infection develops, and attempts to save the cranioplasty usually fail It is wiser to remove the plate, allow the wound to heal completely, and repeat the cranioplasty at a later date. In some cases, infection after cranioplasty occurs if the plate has been applied too soon after healing of an infected or contaminated wound.

Tantalum bends easily on impact a plate which has bent inward as a result of a blow can be removed, recontoured, and replaced (Fig 96) Patients with a tantalum cranioplasty should be advised to cover their heads when they are in the sun because the plate absorbs heat, it has been suggested that this may be a source of headaches, but we have not found it to be so.

The so-called dead space between plate and dura disappears rapidly as the dura grows through the holes in the tantalum to become continuous with the subcutaneous and the pericranial connective tissue

Methyl Methacrylate Cranioplasty (Fig. 100)

Methyl methacrylate is a plastics material For use in cranioplasty, it is supplied in sterile form as a polymeric powder and a monomeric liquid The proportion of liquid to powder that must be used depends on the room temperature, if the room is warm, more liquid than powder is needed Its advantages over tantalum are (*1*) It is radiolucent, so that structures are clearly visualized in pneumoencephalography and other diagnostic procedures (*2*) Screws are not necessary to secure it in place, a distinct advantage in the frontal sinus area (*3*) It permits a full-thickness repair of a defect (*4*) It is easily available for use in the operating room if an unexpected defect is found at operation (*5*) There is no evidence that the material is carcinogenic in man In infants and children, however, tantalum cranioplasty, which permits bone growth underneath the plate, is a better procedure The full-thickness plastic repair may be displaced upward by the growing bone

To prepare the material for a cranioplasty, the powder is placed in a stainless steel basin, the liquid is poured over it (Fig 100*A*, *b*[1]), and the whole stirred continuously with a brain spatula until it acquires a somewhat doughy, adhesive consistency, at which point the mixing is continued under cold, sterile water (Fig 100*A*, *b*[2]) The mass is then taken into the gloved hand and worked as a doughy mass, still under water, it must be fairly pliable for shaping, but not rubbery. The mass is rolled out with a roller to the thickness of the defect to be filled (Fig 100*A*, *b*[3]), and shaped with ordinary surgical scissors after a paper pattern of the defect (Fig 100*A*, *b*[4]) If some parts of the defect are deeper than others, as in the midfrontal area, two or more layers of the material can be used (Fig 100*A*, *b*[5]) In the case of a frontal sinus defect, the entire sinus can be filled in, and thus obliterated completely, after the sinus lining has been removed

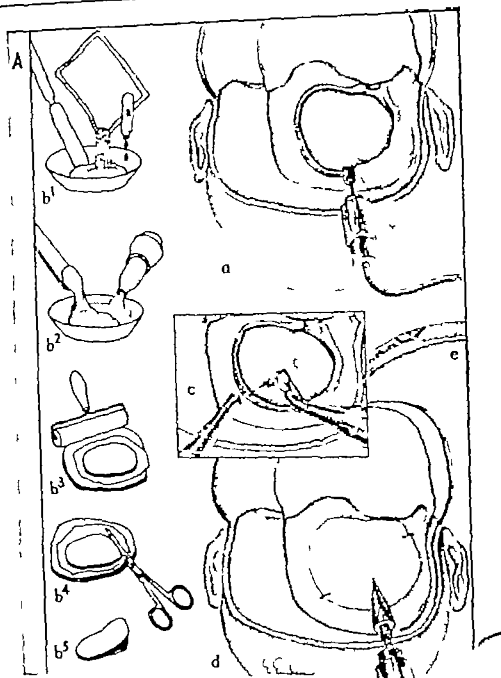


Fig. 100 Methyl methacrylate cranioplasty (*A*) Skull defect exposed (reflected, pericranium separated from dura, bone edge saucerized with b Preparation of methyl methacrylate plate. (*c*) Preparation of bone repair (*d*) Plate wired in place. (*e*) Cross section, showing repair and of plate to skull. (*Continued on next page*)

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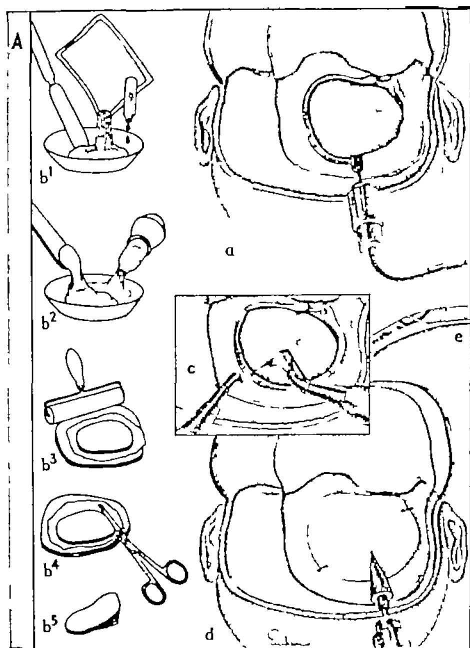


Fig 100 Methyl methacrylate cranioplasty (*A*) Skull defect exposed (*a*) scalp reflected, pericranium separated from dura, bone edge saucerized with burr (*b*) Preparation of methyl methacrylate plate. (*c*) Preparation of bone defect for repair (*d*) Plate wired in place. (*e*) Cross section, showing repair and attachment of plate to skull. (*Continued on next page*)

The so-called dead space between plate and dura disappears rapidly as the dura grows through the holes in the tantalum to become continuous with the subcutaneous and the pericranial connective tissue

Methyl Methacrylate Cranioplasty (Fig. 100)

Methyl methacrylate is a plastics material For use in cranioplasty, it is supplied in sterile form as a polymeric powder and a monomeric liquid The proportion of liquid to powder that must be used depends on the room temperature; if the room is warm, more liquid than powder is needed Its advantages over tantalum are. (*1*) It is radiolucent, so that structures are clearly visualized in pneumoencephalography and other diagnostic procedures (2) Screws are not necessary to secure it in place, a distinct advantage in the frontal sinus area (*3*) It permits a full-thickness repair of a defect (*4*) It is easily available for use in the operating room if an unexpected defect is found at operation (5) There is no evidence that the material is carcinogenic in man In infants and children, however, tantalum cranioplasty, which permits bone growth underneath the plate, is a better procedure The full-thickness plastic repair may be displaced upward by the growing bone.

To prepare the material for a cranioplasty, the powder is placed in a stainless steel basin, the liquid is poured over it (Fig 100*A*, b^1), and the whole stirred continuously with a brain spatula until it acquires a somewhat doughy, adhesive consistency, at which point the mixing is continued under cold, sterile water (Fig 100*A*, b^2) The mass is then taken into the gloved hand and worked as a doughy mass, still under water, it must be fairly pliable for shaping, but not rubbery The mass is rolled out with a roller to the thickness of the defect to be filled (Fig 100*A*, b^3), and shaped with ordinary surgical scissors after a paper pattern of the defect (Fig 100*A*, b^4). If some parts of the defect are deeper than others, as in the midfrontal area, two or more layers of the material can be used (Fig 100*A*, b^5) In the case of a frontal sinus defect, the entire sinus can be filled in, and thus obliterated completely, after the sinus lining has been removed

To repair a skull defect with methyl methacrylate

(1) The scar is excised and all insoluble sutures are removed In frontal wounds, if the original scar is of good cosmetic quality, a coronal incision is advisable.

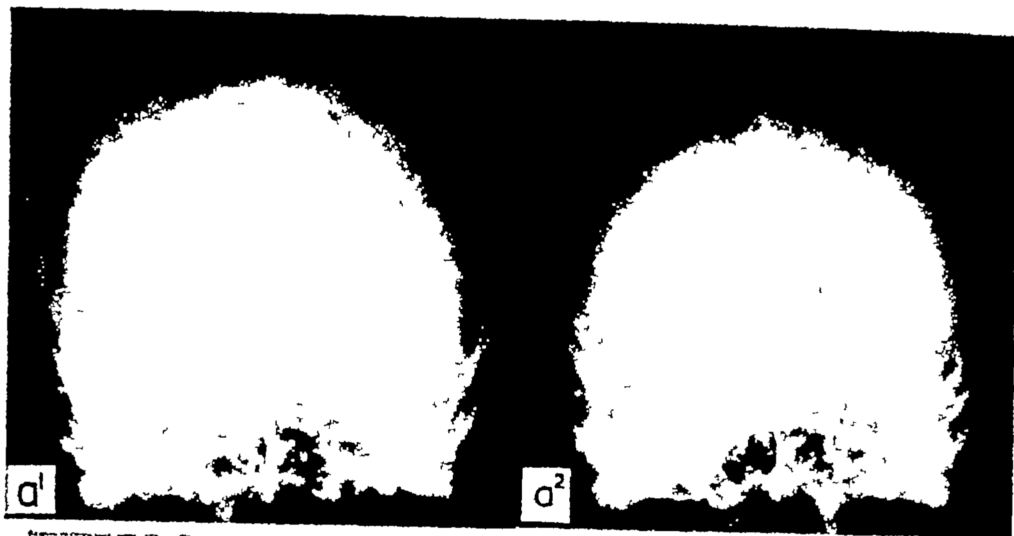
(2) The scalp is reflected, and the pericranium is separated from the bone to form a separate layer, but hinged to one border of the defect (Fig. 100A a), in some cases the latter maneuver may be impossible because the scalp is too thin or an underlying structure, e g the sagittal sinus might be damaged

(3) The edges of the wound or of the skull defect are saucerized out ward by a small burr, held in one hand like a pencil, on a flexible shaft driven from a Luck bone saw (Fig 100A c), the edges of the bone defect should be beveled to provide a tight fit for the cranioplasty

(4) After hemostasis, the methyl methacrylate is prepared and shaped as described above, placed into the defect, and gently pressed so that the edges fit tightly against the borders of the defect If the defect is small, the plastic can be curved with a spatula or the rubber-gloved fingers, contouring the outer surface of the still pliable material For large de fects, several layers of Cottonoid are placed on the dura until the correct thickness and contour are obtained, and the plastic is shaped over this. In the course of shaping and trimming to size, the plastic abruptly be gins to harden and liberates considerable heat, particularly if the operating room is warm, the area should therefore be irrigated with cold normal saline solution. A small excess of the plastic is necessary around the border of the defect to allow for warping

(5) When the material has hardened and set the plate is removed any protruding spots on the inside are smoothed off with an electrically driven burr, the excess around the edges is trimmed off and the edges smoothed with a saw burr

(6) The plate is replaced in the defect, and small holes are drilled in the plate and the skull with a dental head and drill. The holes may be drilled through half the thickness or the entire thickness of bone, depending on the area being repaired and the thickness of the skull.





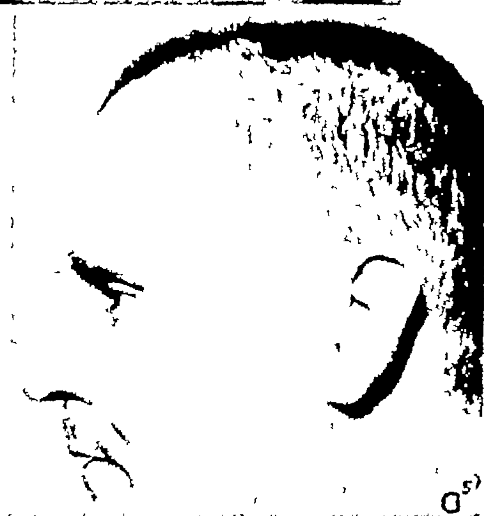


Fig. 100 (*continued*) (*B*) Repair of depressed fracture of frontal sinus. Before and after repair (a^1–a^2), and appearance before (a^3) and after (a^4–a).

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(5) When the material has hardened and set the plate is removed, any protruding spots on the inside are smoothed off with an electrically driven burr the excess around the edges is trimmed off and the edges smoothed with a saw burr

(6) The plate is replaced in the defect and small holes are drilled in the plate and the skull with a dental head and drill The holes may be drilled through half the thickness or the entire thickness of bone, depending on the area being repaired and the thickness of the skull

(7) The plate is wired into place, and the edges again trimmed, if necessary, so that the plate is level with the surrounding bone (Fig 100*A, d*). In repairing a supraorbital ridge, care must be taken to avoid any protrusion of the material downward.

(8) The wound is thoroughly rinsed so as to remove all particles of plastic and bone, then rinsed with a small amount of penicillin solution, and the scalp flap is sutured into place.

(9) A pressure dressing is applied lightly for the first 24 hours

For a large plate over the anterior lateral aspect of the skull, with its curvatures in different directions, a pattern of the defect is first made of metal After preparing and rolling out the plastic to the desired thickness, it is applied to the interior of the metal pattern and the whole is tightly held against the defect Once the plastic has set, cold water is poured over the metal plate, and plastic and metal are easily separated from each other. This technic permits more efficient application of the plastic to large areas and better cosmetic results

Facial Nerve Injury (*Fig. 101*, see also *Fig. 48*)

A peripheral facial paralysis in patients with closed head injury is temporary in about 90 per cent of cases, and improves to some extent in the remaining 10 per cent, in this group, mass movements of the face on volitional attempts are common Treatment at first should be conservative physiotherapy, massage, mild heat, and exercise of the facial muscles The response of the facial muscles to faradic and galvanic stimulation may be used as a gu[illegible] gnosis [illegible] for the need of further treatment If facial fun[illegible] end of 3 months, and there is tearing [illegible] nse of taste is not affected. decompres[illegible] may prove valuable [illegible] raft [illegible] lly damaged Pl[illegible] and a bucca[illegible] the side, may b[illegible]

Open injuries by shell fragments or bullets may be so extensive as to make repair of any part of the facial nerve impossible. In some of these cases, which cannot be treated by direct nerve anastomoses, nerve grafts have been successfully used, it is emphasized that repair of the

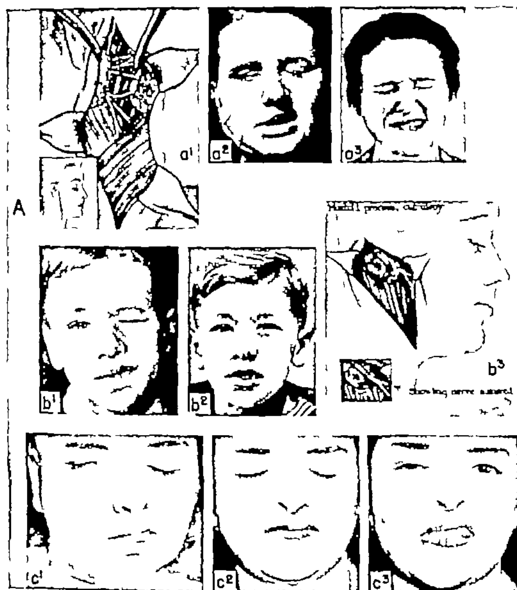


Fig 101 Facial nerve injuries (*A*) All branches of facial nerve and parotid gland sectioned (*a*) each branch was sutured, and complete recovery followed (*a*³) (*b*) Recovery from facial paralysis after suture of facial nerve as it emerged from facial canal (*b*¹) (*c*) Facial paralysis due to section of facial nerve just outside facial canal complicating parotid fistula complete recevorey after 9 months (*Continued on next page*)

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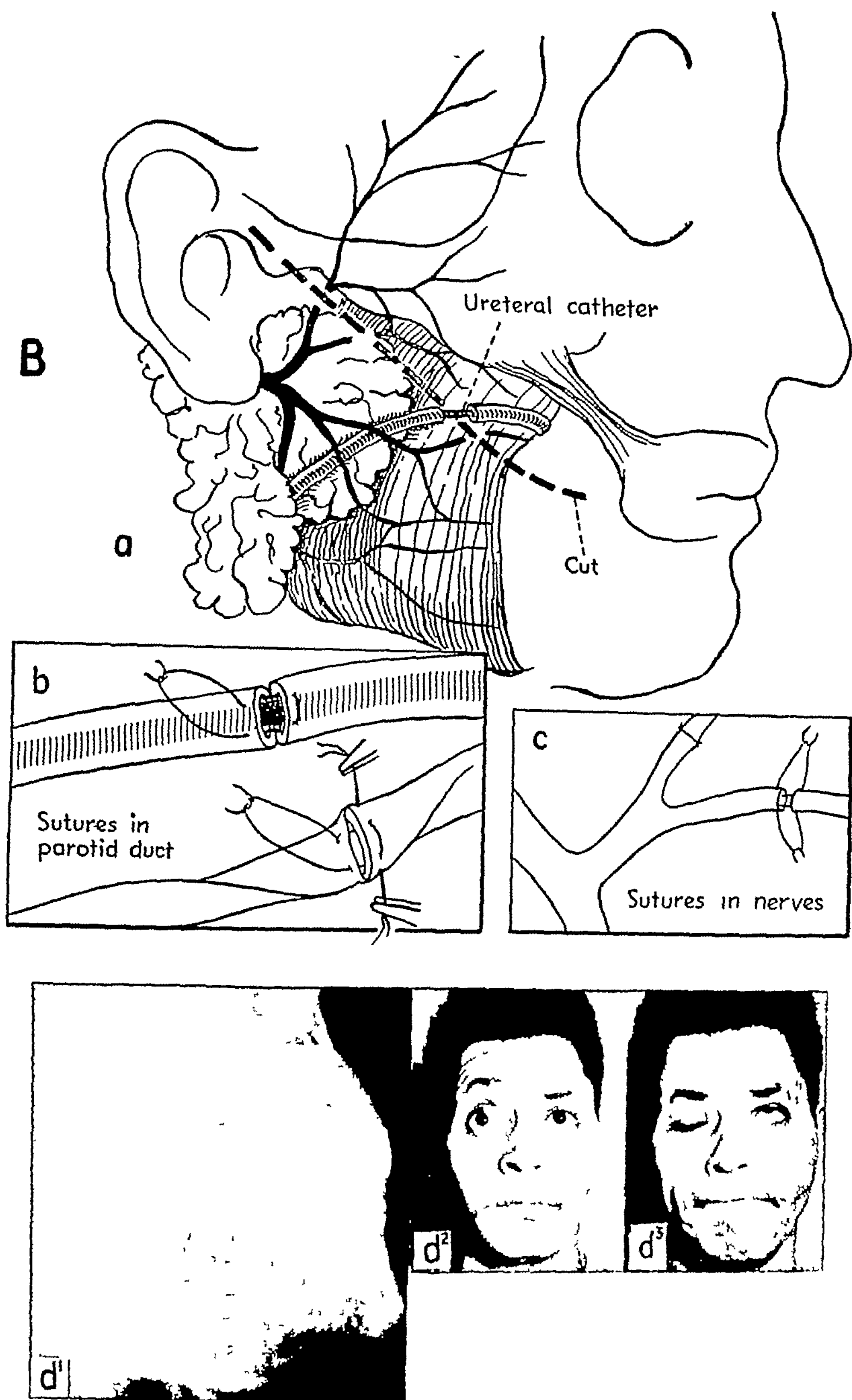


Fig 101 (*continued*) (*B*) Management of section of facial nerve and parotid duct (*a*) Ureteral catheter *in situ* for suture of parotid duct (*b–c*) Suture of duct and of nerves within 24 hours after injury (*d*) Roentgenogram and appearance of patient with involvement of upper portion of facial nerve and section of parotid duct Patient recovered

branches of the facial nerve is more easily accomplished immediately after the facial injury than after the wound has healed[18, 83] In the latter type of case, a fair restitution of function has been obtained by the use of nerve grafts.[18, 83] A graft may also be tried when a major part of the nerve near or in the facial canal has been destroyed

The possibility of complete recovery of function is good when the facial nerve is involved in stab wounds or when the nerve trunk (or its branches) is lacerated, in such cases the nerve can usually be anastamosed (Fig 101*A b-c*) For success of treatment, however it is most important that exploration and suture of the nerve, particularly of the branches distal to the pes anserinus, be undertaken immediately after injury (Fig 101*A a*), late after an injury which has sectioned the branches, scar tissue makes their identification impossible. If the parotid duct is sectioned, it may be sutured around an inlying ureteral catheter (Fig 101 *B, a d*), which is removed after 1 week. If there is a complicating parotid fistula, roentgen ray irradiation of the parotid gland helps to prepare the wound for exploration by stopping secretions.

For irreparable injury of the central portions of the facial nerve, *i.e* the portions lying in the internal acoustic meatus, a facial-hypoglossal nerves anastomosis, or a facial-spinal accessory nerves anastomosis (*see* Fig 48*e*), is a useful procedure. This operation was first performed by Ballance[4] in 1895 A certain amount of tone develops in the facial muscles which encourages symmetry when the face is at rest, voluntary facial movements, however, depend on associated shoulder or tongue movements. Some patients become adept in the use of such movements to effect expressive facial changes.

Vascular Lesions and Hematomas

Patients with head injury may have vascular lesions without signs of massive bleeding The intracranial anatomic relationships may prevent bleeding from a torn blood vessel or a bone fragment may compress a torn sinus and thereby temporarily stop the bleeding On the other hand a fracture or depression may initiate a massive intracranial

hemorrhage, and, in some cases, traumatic hemorrhage may be simulated by occlusive cerebrovascular disease Careful preoperative evaluation with the proper diagnostic technics (*see* Chapter IV) is therefore obligatory in every case

Carotid Artery–Cavernous Sinus Fistula

In most cases, the treatment is surgical The common carotid, the internal carotid, and the intracranial portion of the internal carotid arteries are successively ligated, as may be indicated [15] Usually, ligation of the common or the internal carotid artery in the neck suffices for cure If treatment has not been too long delayed, the eyeball can be saved, although not always with normal vision In 1 of our cases, in which ligation of both the common and internal carotid arteries failed to effect a cure, the introduction of a long piece of muscle into the internal carotid sealed the tear and led to complete subsidence of the exophthalmos In the occasional case of extremely severe exophthalmos with corneal ulceration, the eye may have to be enucleated

A number of workers have reported on bilateral carotid artery ligation, which they found to be necessary in unusual circumstances [33, 34, 52]

Dural Sinus Injuries (see Figs 53-54)

Any of the venous sinuses may be injured, but most frequently it is the sagittal and transverse sinuses With a comminuted fracture of the petrous bone, the superior and inferior petrosal sinuses are apt to be injured With closed or open depressed fractures of the vertex in the midline, the sagittal sinus may be involved A tear of the lateral sinus may occur with comminution and depression of the occipital squama Fractures of the base may involve the body of the sphenoid, and cause varying degrees of cavernous sinus injury In penetrating head injuries, any of the dural sinuses may be injured [54] In civilian practice, the common dural sinus injuries include involvement of the superior sagittal, the lateral, the superior petrosal, and the sphenoparietal sinuses

Bleeding from injured dural vessels may be extradural only, both extradural and intradural, or intradural only In some cases of a tear of a

dural vessel due to a depressed fracture or a penetrating head injury, active bleeding starts only when the wound is debrided and the depressed bone fragment or some foreign material which has plugged the tear is removed. One must therefore be prepared for hemorrhage from a torn sinus when such a head injury without obvious extradural or intradural hemorrhage is being debrided Adequate exposure of the operative area before beginning to remove bone fragments is essential, since this permits whatever manipulations become necessary A piece of muscle, obtained preoperatively and held in readiness, may prove valuable if an extensive laceration is exposed in the course of the debridement. Minor tears may be sutured, and extradural bleeding points are controlled by electrocautery, silver clips, or Gelfoam, as indicated electrocautery should not be used on the superior longitudinal and transverse sinuses. The sinus must be compressed throughout the operative procedure to prevent air embolism, which may be fatal

If a venous sinus is so extensively damaged that it must be ligated, the area of its drainage may become infarcted This is particularly true when the posterior half of the sagittal sinus has been ligated It has been found that if the wound is reopened 5 to 10 days later and the infarcted area debrided, the maneuver may not only be lifesaving but may also result in the return of some function.[54]

Sagittal Sinus (Fig 102; see also Figs. 53-54)

Only in its anterior third may this sinus be ligated if ligation is deemed necessary A minor tear in the sinus wall is repaired by a piece of Gelfoam or muscle, held in place with several interrupted silk sutures bridging the material from the dura on one side to the other (Fig 102*A* *a*) A small longitudinal tear is repaired by incising the dura on either side of the torn sinus to relieve tension and suturing the tear with interrupted silk sutures, then closing the dura by suture or repair, as may be necessary (Fig 102*A* *b*) A large tear in the anterior third is closed by double ligation (Fig 102*A* *c*) To repair a tear in the lateral aspect of the sinus, it is adequately exposed by incising the dura and elevating the edge of the dura, after removing the clotted blood,

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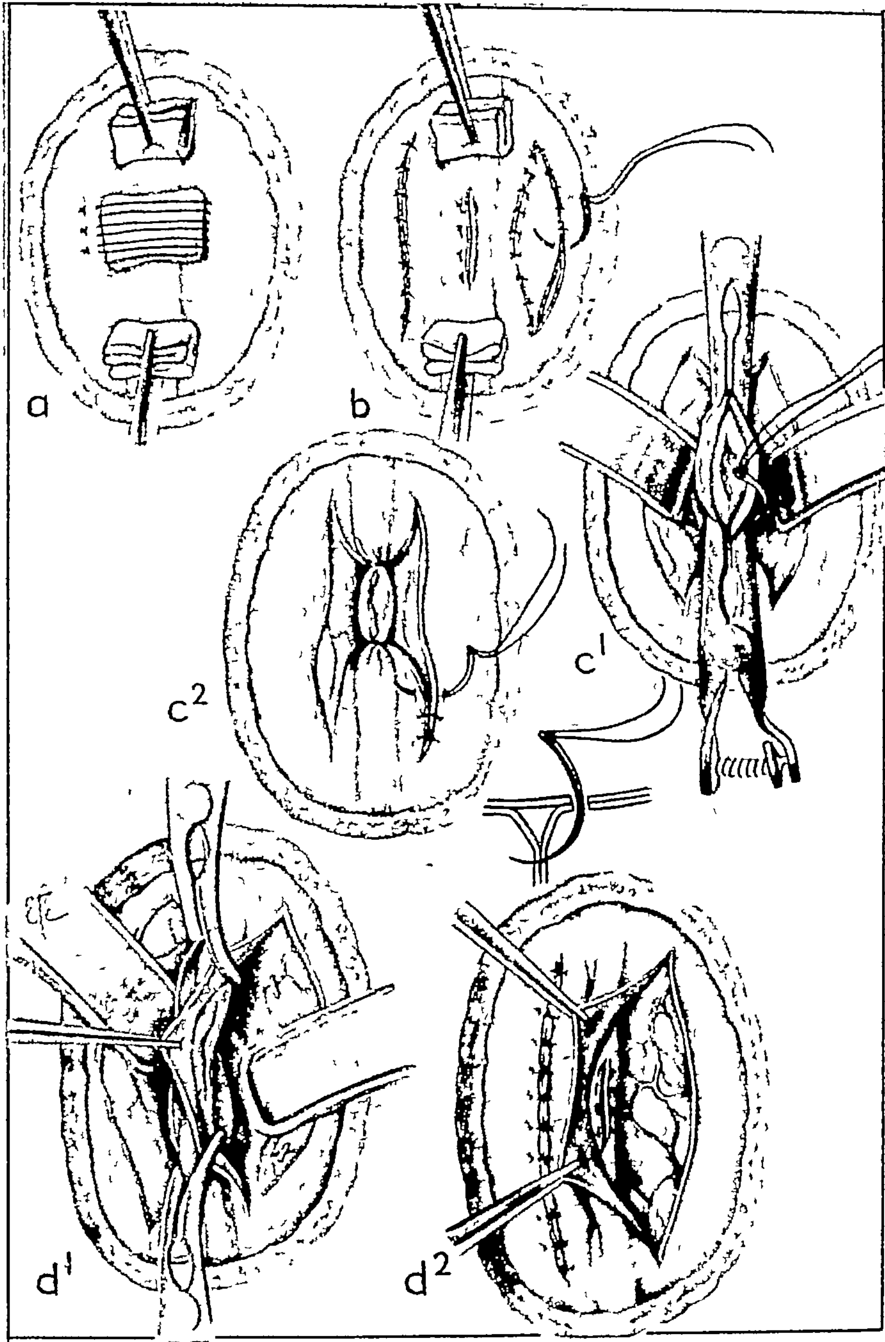


Fig. 102. Management of sagittal sinus tears (*A*) Repair of small tear (*a*) by Gelfoam or a piece of muscle held in place by several bridging sutures (*b*) Repair of small longitudinal tear (*c*) Double ligation for tear of anterior third of sinus. (*d*) Repair of tear in lateral aspect of sinus.

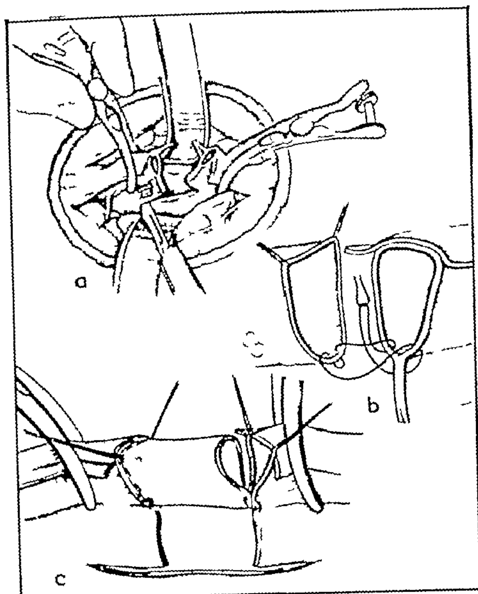


Fig 102 (*continued*) (*B*) Repair by vein graft. (*a*) Control of bleeding by arterial clamps. (*b*) Mobilization of free ends of sinus by incising falx cerebri and dura on either side of tear (*c*) Suture of vein graft in place.

hemorrhage is controlled by 2 arterial clamps, the tear is closed with everting sutures (Fig 102*A* *d*), if necessary, Gelfoam may be placed over the area. A serious injury which destroys the continuity of the vessel may be repaired by vein graft. This may be a particularly valuable technic in the commonly massive intracranial damage of war injuries. After adequate exposure arterial clamps are applied to control

bleeding, the dura on both sides of the sinus and the vessel's attachments to the falx are incised carefully in order to relax the vessel ends (Fig 102*B, a*), the ends of the sinus are mobilized; and a vein graft is sutured in place by interrupted or continuous everting sutures (Fig 102*B, c*) In the Korean War, double ligation instead of vein graft[54] was used, but there were serious sequelae.

Transverse or Lateral Sinus (see Fig. 54B)

Tears of this sinus may be doubly ligated There are no serious complications, papilledema may occur, but is temporary Electrocautery of bleeding points is inadvisable, except for veins which empty into the sinus

Superior Petrosal and Sphenoparietal Sinuses

If the superior petrosal sinus is torn by a comminuted fracture of the petrous pyramid (*see* Fig 17*B*), bleeding may be controlled by electrocoagulation or by silver clips The sphenoparietal sinus and its tributaries may be torn by depressed fractures involving the posterior border of the lesser wing of the sphenoid, electrocoagulation effectively stops bleeding

Piarachnoid Adhesions after Subarachnoid Hemorrhage

When the adhesions are extensive and cause obstructive hydrocephalus, particularly in infants and young children, it may be advisable to consider resort to a by-passing operation, such as the Torkildsen[80a] procedure, in which a passage is created between the lateral ventricle, the cisterna magna, and the subarachnoid space of the spinal canal In some cases, the use of the Holter[78a] valve to afford passage of excess cerebrospinal fluid into the venous system may be valuable

Extradural Hematoma (Figs. 103-104)

Active bleeding from the middle meningeal artery or its branches, and the middle meningeal veins, the main source of extradural hematoma, is infrequently encountered on operation, for the vessels have usually

thrombosed by the time surgical intervention is undertaken The exact location of the various branches of the artery has been known for some time,[43, 66, 70] so that trephination to locate the hematoma can be done accurately The earlier practice of ligating the common and external carotid arteries for hemorrhage of the middle meningeal artery[33, 52, 66] has long been abandoned but recently Raney and co-workers[7] have suggested it as a preliminary maneuver until the patient reaches better surgical facilities.

Preliminary diagnostic procedures to permit the localization of the clot may be desirable in most cases. In a few diagnostic burr openings may be made along a fracture line The usual procedures now used for extradural hematoma are removal of the clot through a subtemporal de compression or by an osteoplastic flap Cushing[14] preferred the former, Krause[41] suggested the latter

Prompt surgical treatment is mandatory in *infants and children* The preparation for surgery should include the availability of blood for transfusion in the operating room, a cut-down in an ankle vein should be made before the operation, with a cannula in place, so as to be ready for the transfusion.

Subtemporal Decompression for Extradural Hematoma (Fig 103)

For a hematoma in the temporal area (*1* in Figure 103*a*), the skin and the temporal muscle are incised (Fig 103*b*) The skull is then opened near the fracture line close to the base and the zygomatic arch, and the opening is enlarged to the size of a silver dollar or larger, this exposes the hematoma (Fig 103*c*) The clot is removed by suction or by a brain spatula. Hemostasis must be thorough, as soon as bleeding points appear they are controlled with a tenting stitch, using silk sutures (Fig 103*d*-*e*) electrocautery may be used, if necessary The dura is opened and the subdural area is inspected for subdural cerebrospinal fluid collection or hematoma it is then closed with interrupted silk sutures. A drain is usually inserted at the lower end of the skull opening (Fig 103*e*) and the wound is closed in layers with interrupted silk sutures (Fig. 103*f*-*h*)

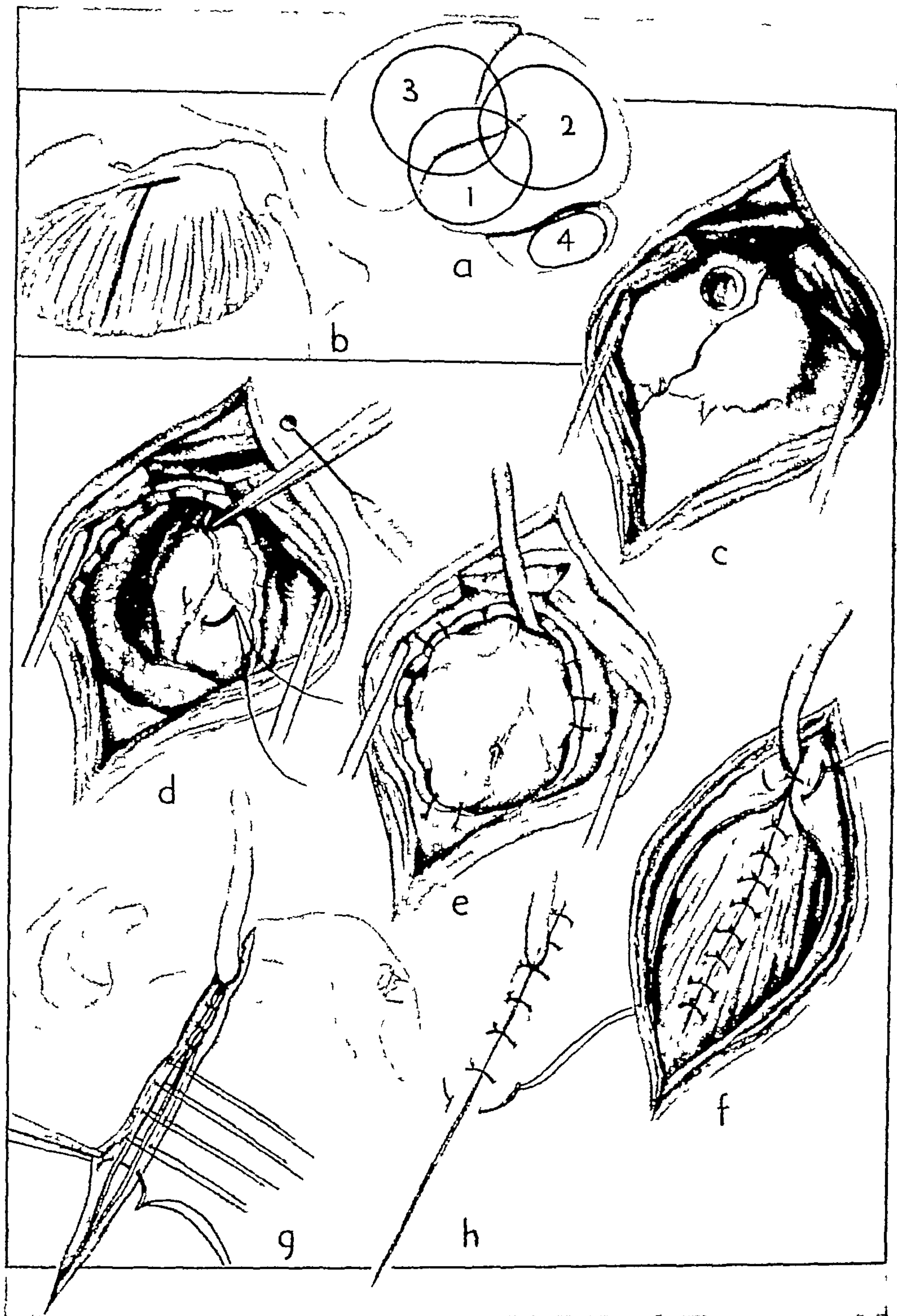


Fig. 103 Management of extradural hematoma by subtemporal decompression (*a*) Most common sites of extradural hematoma. (*b*) Incision of temporal muscle (*c*) Opening in skull (*d*) Skull opening enlarged, exposing hematoma, suture of middle meningeal artery and application of electrocautery. (*e*) Dura closed with tenting stitches, drain at lower end. (*f–h*) Wound closed in layers.

If preliminary burr openings have been made to locate the extra dural hematoma, they are incorporated in the subtemporal decompression opening These burr openings are usually made in a position just above and in front of the ear, as well as just above and behind the ear on the affected side.

Osteoplastic Bone Flap (Fig 104)

This is the method of choice for a hematoma beyond the confines of the temporal bone and muscle. Frontal and parietal hematomas are well visualized by pneumoencephalography and angiography, and the decision with regard to operative procedure can be made preoperatively A skin flap is turned for a frontal osteoplastic craniotomy the incision extending from the middle of the forehead to the temporal area in front of the ear A horseshoe incision is used for the parietotemporal area, with the mouth of the horseshoe around the ear on the affected side A more posteriorly placed horseshoe incision may be used to explore the occipital portions of the head After the skin flap is turned several openings are made with a hand or a mechanical drill, the bone between the burr holes is sawed with a Gigli saw, and the bone flap is turned, the base of the flap being left attached to the temporal muscle. After the dura is exposed, the hematoma comes into view, the clot is removed by suction or with a brain spatula. A subdural hematoma and/or an accumulation of cerebrospinal fluid are frequently associated with the extradural hematoma. Therefore, after the extradural hematoma is removed, the dura is incised for subdural exploration, particularly if the dura is tense or bluish. If the exposed brain seems to bulge, its depth may be explored with a brain cannula for an intra cerebral hematoma. A coexistent subdural hematoma or hygroma, or an intracerebral clot, is evacuated, all bleeding points are secured and the dura is closed and tented with interrupted silk sutures (Fig 104*d*'), tenting of the dura is extremely important since evacuation of the extradural clot usually leaves a large dead space between dura and bone which may encourage secondary hemorrhage If the dural opening is ex-

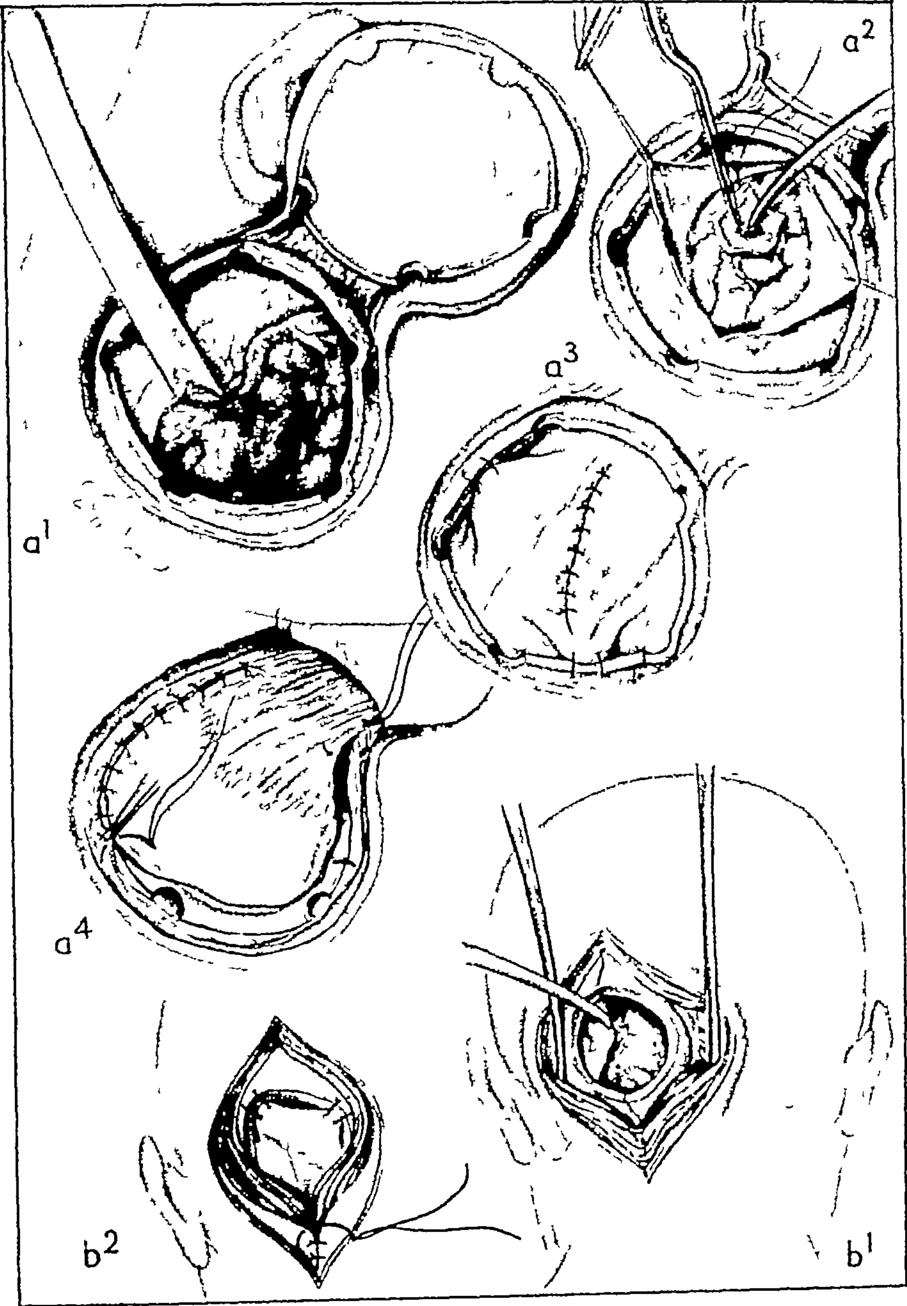


Fig 104 Management of extradural hematoma by osteoplastic flap (*a*) Management of parietal hematoma, (a^1) exposure and removal with spatula, (a^2) dura incised, and subdural hygroma removed by suction over Cottonoid, (a^3) dural incision closed and dura tented, (a^4) bone flap sutured (*b*) Exposure of hematoma in posterior fossa and closure of wound

tensive, a complete incision, so as to create a free graft when the dural sac is closed, may be preferable. The bone flap is replaced and fastened with 2 or 3 wire sutures the muscle and skin incisions are then closed in layers with interrupted silk sutures. A drain, removed at the end of 18 to 24 hours, may be used for drainage of the extradural and epicranial regions of the wound

The management of an extradural hematoma in the posterior fossa over the left occipital lobe is illustrated in Figure 104*b* A large opening is made by a paramedian incision. Complete hemostasis is insured by ligation or careful cautery of the occipital vessels if the exposure is more lateral, bleeding from the auricular artery and vein must be controlled. The hematoma is removed, and bleeding is controlled by Gelfoam held in place over the area, the source of the bleeding may be a torcular vein or a communicating vein between the scalp and the transverse sinus. If there is evidence of increased intradural pressure the dura is opened and the cerebellum is tapped for a subdural or intracerebellar collection. The dura is tented to prevent postoperative secondary hemorrhage and the wound is then closed in layers with interrupted silk sutures, after complete hemostasis.

Subdural Hematoma and Hygroma (Figs. 105-106)

The principle of management of a subdural hematoma is evacuation of the collection It is important to bear in mind that the earlier surgical intervention is necessary, the poorer the prognosis The type of procedure depends, to some extent, on whether the collection is solid (as it is soon after trauma), or liquid, which in our experience is true of more than half the cases The possibility that there may be a coexisting intracerebral hematoma, or that the subdural hematoma may be a manifestation of an intracerebral hematoma draining into the subdural area, should also be kept in mind

For a liquid hematoma 2 bilateral burr openings on both sides of the midline in the frontal area (over the coronal suture, about 3½ cm. on either side of the midline), and 2 in the temporoparietal area (just above

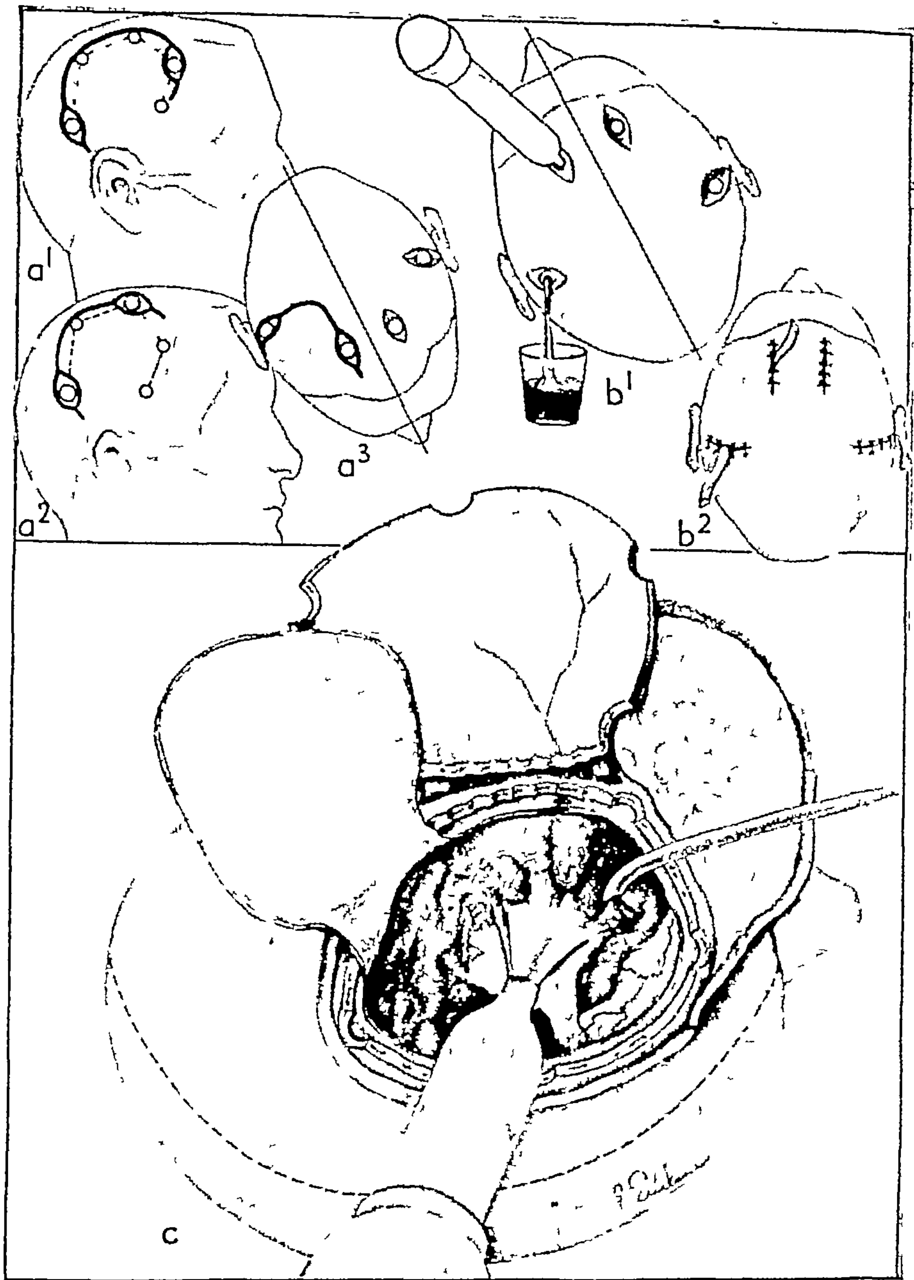


Fig 105 Management of subdural hematoma (*a*) Trephine openings and incisions for bone flap (*b*) Drainage of liquid hematoma, and closure, with drains, of trephine openings (*c*) Solid hematoma exposed by bone flap.

and posterior to the ear) are made and the clot is drained with the help of saline solution through a rubber catheter (Fig 105*b*') Escape of the saline solution must be facilitated by careful manipulation of the catheter or by depressing the brain with a blunt instrument, since trapped fluid compresses the brain. The dura overlying the clot is coagulated and attached to the bone border of the burr opening, this prevents formation of another hematoma from a bleeding dural edge or from separation of dura from bone. The incisions are then closed, with a drain(s) on the side of the clot (Fig 105*b*') the drain should be removed in about 24 hours. Burr openings may also be so placed that they may be used to turn a bone flap (Fig 105*a*) After incision, a flap of skin is turned and then a small bone flap by utilizing the initial 2 openings in addition to others (Fig 105*c*) The clotted material is carefully sucked and washed away so as not to injure the cerebral surface. Hemostasis is important. In closing the wound, tenting of the dura is advisable The presence or absence of an intracerebral clot should be established by preliminary angiography

For the *subacute* subdural hematoma, multiple burr openings are usually made, so that the type of clot can be identified and its drainage facilitated A liquid hematoma is drained in the same manner as the acute liquid hematoma A small osteoplastic flap must be turned for a hematoma which is largely or completely solid and has a formed membrane (*Fig. 106d'*) *On opening the dura, the outer membrane of* the hematoma can be seen (held in forceps in Figure 106*d*') as the clot is removed, its thin inner membrane over the cerebral surface becomes visible. Complete hemostasis is essential in all procedures for subdural hematoma. Tenting of the dura, using silk sutures is advisable (Fig 106*d*') A free dural graft will prevent the possibility of bleeding from dural vessels.

A *chronic* subdural hematoma, if liquid, can be treated by 2 burr openings to provide counterdrainage.[40] In every case of chronic subdural hematoma the exploration must be done bilaterally

A chronic or subacute hematoma particularly if large, may compress

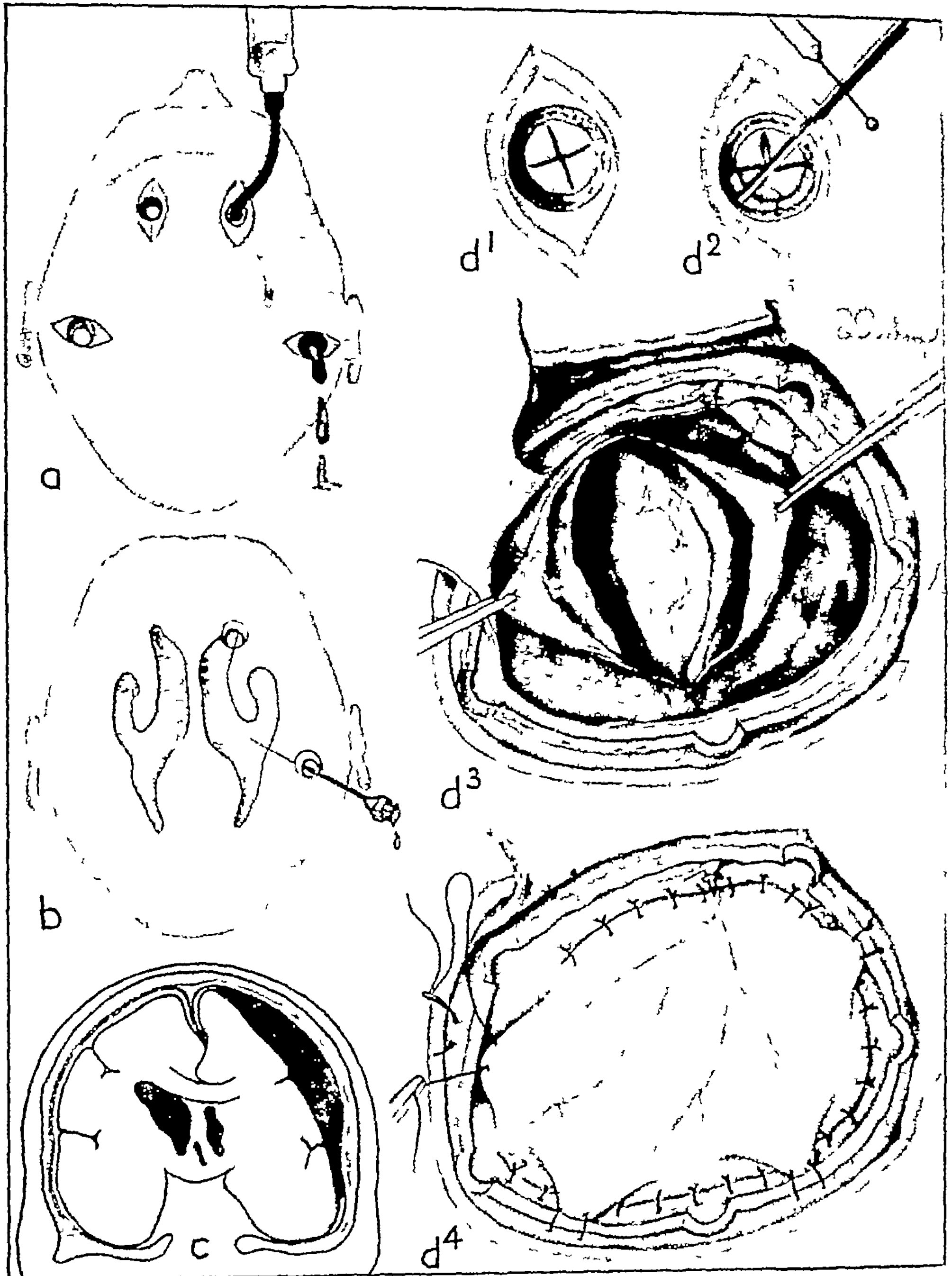


Fig 106 Management of subacute and chronic subdural hematomas (*a*) Liquid hematoma exposed and drained (*b*) Injection of air or saline into ventricle (*c*) Extradural hematoma compressing cortex and causing ventricular shift (*d*) Management of solid hematoma, burr opening and electrocoagulation of dura (d^1–d^2), bone flap turned to expose hematoma (d^3), and dura tented (d^4).

the cortex, or cause a shift of the ventricular cavities (Fig 106c) or extensive cerebral depression. Such defects may remain even after the clot is removed In such cases, a needle can be introduced into the lateral ventricle on the side of the lesion and air or saline injected (Fig 106b) Air or saline has also been injected into the spinal subarachnoid space for the same purpose, in some cases.[46]

Management of subdural hematoma or hygroma in *infants and children* calls for special care and measures. In the infant particularly the cerebral surface is easily bruised, and sudden changes in intracranial pressure, such as result from burr openings may cause cerebral herniation through the opening The management is largely governed by the pathologic characteristics of the lesion Burr openings are the preferred method for establishing these characteristics. If they reveal a membrane enclosing the hematoma the openings are utilized for a small bone flap bilaterally if necessary, so that as much of the membrane can be removed as possible. Occasionally repeated drainage of the subdural fluid results in symptomatic recovery If the cortex herniates through the burr openings it may be bruised, to prevent this, the cerebral surface is gently compressed with a blunt instrument covered with Cottonoid which releases more of the clotted material for escape. Another useful maneuver is the placing of a small Cottonoid pledget soaked in saline in the subdural space the pledget soaks up blood and liquid which is then aspirated by suction. After several aspirations, the brain recedes and more of the clot may escape.

A subdural *hygroma* (cerebrospinal fluid collection) is treated by drainage through bilateral burr openings, and removal of the membrane if one has formed through a small bone flap When the dura is opened the clear straw-colored or xanthochromic fluid usually spurts out, the forcefulness depending on the intracranial pressure and the size of the dural opening The opening may be enlarged and the cerebral surface carefully compressed by a blunt instrument covered with Cottonoid this may result in the escape of additional fluid Since trapped fluid tends to crowd the cerebral tissue about the opening care

ful compression of the brain may drain large quantities of fluid A small pledget of Cottonoid introduced into the subdural space may absorb escaping fluid as it is sucked away; this helps to shrink the brain down

Although the procedure is simple, the mortality rate of patients with subdural collections of cerebrospinal fluid is 30 per cent. This is undoubtedly the result of its frequent association with severe intracranial damage

In infants, traumatic hydrocephalus and persistent subdural accumulation of cerebrospinal fluid can be treated by inserting a Holter valve[78a] to connect the ventricular or subdural area with the jugular vein and the superior vena cava This insures the outflow of fluid at pressures above 50 mm of water. The valve lies just behind and below the ear, it can therefore be tested to check blockage either in the cranial or the venous outlet This method has been found to be far superior to the other shunting procedures from the ventricle or spine to the abdomen or ureter, in hydrocephalus

Intracerebral and Intracerebellar Hematomas (Fig. 107)

The most common site of an intracerebral hematoma is the temporal lobe Parietal, occipital, and cerebellar hematomas are next in frequency, in that order Angiography is very useful in establishing the diagnosis of intracerebral hematoma, particularly in the frontal and temporal areas Results of treatment are poor, the mortality rate, in our experience, being 55 5 per cent.

For a hematoma in the temporal region, subtemporal decompression is a good procedure, the trephine opening being placed over the temporal lobe just above the zygomatic arch and in front of the ear (Fig 107*c*). The opening is enlarged until it is about the size of a silver dollar, and a circular flap of dura is then reflected (Fig 107*c*[2]) The bulging, discolored brain tissue is sucked away (Fig. 107*c*[3]) or removed by electrocautery, thus exposing the clot. This, too, is sucked away and the cavity is washed out with saline The dura is then replaced and

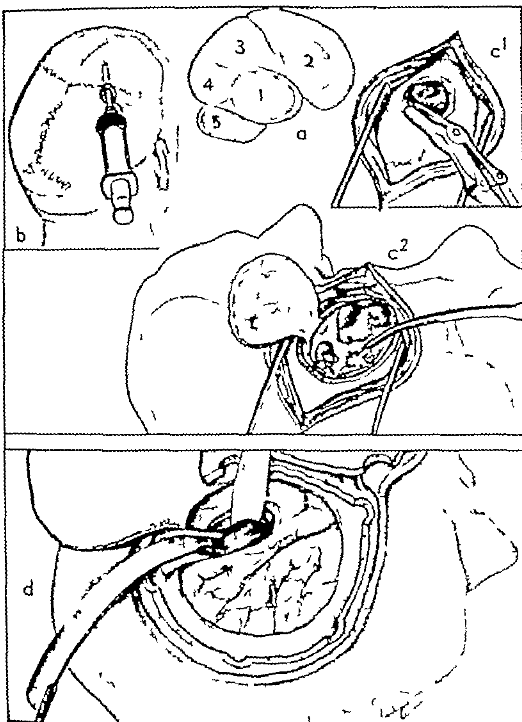


Fig 107 Management of intracerebral hematoma. (*a*) Possible sites of hematoma. (*b*) Frontal lobe hematoma removal by suction. (*c*) Temporal craniotomy technic for temporal lobe hematoma. (*d*) Bone flap technic for parietotemporal hematoma.

sutured in place with interrupted silk sutures We find this method preferable to aspirating the clot through a small burr opening

A frontal lobe intracerebral hematoma can be removed by suction with a syringe introduced through a burr opening (Fig. 107*b*) A bone flap may be preferable, if the patient's condition permits

A parietotemporal intracerebral clot (Fig 107*d*) is exposed by means of a bone flap, the tissue overlying the clot is carefully excised by electrocautery and suction, the cavity is entered, the clot is adequately visualized by means of lighted retractors, and is removed with suction and brain spoons Hemostasis should be complete The dura is then replaced and sutured with interrupted silk sutures, the bone is replaced and held in position with wire sutures, and the scalp is closed in layers with interrupted silk sutures

Intracerebellar hematomas may cause lateralizing signs, in such cases, a unilateral cerebellar exploration may be undertaken Bilateral suboccipital exploration may be preferable in some cases Through a midline incision on the back of the head and neck, extending from above the external occipital protuberance to the fourth cervical spinous process, the occipital squama and arch of the atlas may be exposed unilaterally or bilaterally For bilateral exploration, the occipital squama is removed from mastoid to mastoid and from the superior nuchal line to the foramen magnum, for unilateral exposure, the opening should extend from the superior nuchal line to the foramen magnum and from the mastoid to beyond the midline The dura is opened by a *T* incision for bilateral exposure, and by an upside down and reversed hockey incision for unilateral exposure The cerebellum is tapped with a blunt exploring needle, if a clot is present, the cerebellar cortex may be removed by suction and electrocautery so as to uncap the cavity of the clot The clot is then washed and sucked away After complete hemostasis, the dura is closed with interrupted silk sutures, and the muscles and skin are closed in layers For posterior fossa explorations, the posterior arch of the atlas should also be removed. A burr opening in the parieto-occipital area near the midline (8 cm. above the nuchal line,

3 cm. from the midline) for ventricular drainage may help to control intracranial pressure.

Pneumocephalus[12, 22, 30, 71]

For the effective management of pneumocephalus, the site of the fistulous tract, whether it is patent or not, and whether the condition is progressive must be ascertained

Treatment of a *cranioaural fistula* indicated by cerebrospinal fluid or bloody otorrhea, is usually conservative. Prophylaxis with antibiotics, strict bed rest in a semi Fowler position for 3 or 4 weeks, guarding the patient against "head colds, and avoidance of straining and coughing spells, are the principal measures. Large enemas are contraindicated Frequent roentgenography during this period may be useful in showing the response to treatment. If the accumulation of air continues to increase, operative intervention is probably necessary, but if successive roentgenograms show that the air is slowly disappearing conservative treatment may be continued.

Whatever the cause of the pneumocephalus, every case must be evaluated individually Since the introduction of the chemotherapeutic and antibiotic agents, operative repair of the fistula would seem to be the safest and most advisable method of dealing with pneumocephalus, particularly in those cases in which the pneumocephalus occurs some time after injury

Treatment of an acute pneumocephalus complicating an open depressed fracture in the frontal sinus area or elsewhere is surgical. So too, is the treatment of cerebrospinal fluid rhinorrhea with or without pneumocephalus, complicating a comminuted fracture of the posterior wall of the frontal sinus.[31]

In almost all cases of pneumocephalus appearing months or years after a head injury, there is a cranionasal fistula which is a constant menace as a source of infection and of repeated attacks of meningitis. Repair of the fistulous tract is therefore mandatory and especially if

the pneumocephalus is an intracerebral one. Of 8 patients so treated by us, 7 recovered, and the literature contains many reports of successful treatment.[11, 12, 23, 56, 80] Nevertheless, good results have also been obtained with conservative treatment.[5, 7, 20, 43, 50, 64]

After complete exposure of the area, a cranionasal or cranioaural fistula is best repaired by a fascial transplant, or by mattress sutures if the tract is small. Fascia lata obtained from the thigh may be used as a graft inside the dural opening if the torn area is extensive. In every case, the fistulous tract must be completely closed. Gelfoam or plugs of muscle cannot be depended on for complete closure. Before and after the operative procedure, adequate doses of the sulfonamides and antibiotics are administered.

Otitis Media and Mastoiditis[68]

The presence of either condition should be noted early and appropriate treatment instituted. Mastoid suppuration which persists despite drug therapy calls for a mastoidectomy. Suppuration in the air cells of the petrous bone may require surgical exenteration of the involved cells.

Osteomyelitis

In head injuries with extensive scalp destruction, the skull should be covered with dressings kept wet with boric acid solution until granulation tissue has formed. This will usually prevent an osteomyelitis due to drying out of the bone. To speed the granulation process, the outer table of the skull in the involved area may be removed, or small, closely spaced holes may be made into the diploe with a burr; granulation tissue soon grows out of the diploe to cover the entire exposed area. If bone and overlying scalp are completely destroyed, the entire area of exposed bone should be excised, including a border under the skin edge (*see* Fig. 85*c*). Granulation tissue then forms over the dura.

If the osteomyelitis is a result of inadequate debridement or of lack of

treatment of an open depressed skull fracture, the osteomyelitic area is excised and the wound is irrigated with penicillin solution. Usually foreign matter, hair dirt and other debris are found between the bone fragments, as well as some purulent matter extradurally and sequestration of bone. Occasionally, the osteomyelitic process extends beyond the area of injury Wide excision is then necessary for cure.

Osteomyelitis of the bones around the paranasal sinuses due to patent cranionasal fistula is treated by complete excision of the involved area, antibiotics, and sulfonamides. If smear and culture studies isolate an organism, its sensitivity to antibiotics should be ascertained, and therapy altered accordingly

Intracranial Abscess (see Figs 66 67)

Although the presence of intracranial abscess was recognized as far back as the sixteenth century treatment was for a long time considered hopeless. Gross,[7] in his review of the subject in 1873, stated that evacuation of a brain abscess was invariably unsuccessful Before the days of the sulfonamides and antibiotics, successful treatment depended on removal of the suppurating mass without contaminating the surrounding uninfected tissues. In recent years, with the use of antibiotics and sulfonamides, the treatment of intracranial suppuration has become fairly well standardized

Most extradural abscesses can be evacuated without opening the dura. Usually, the craniotomy should be extensive enough to remove all infected bone. Granulation tissue on the dura should be debrided completely In the presence of an associated intradural mass, drainage or removal of the suppuration should be accomplished as described below The exposed extradural area is washed with an aqueous solution of 500,000 units of penicillin solution in 10 cc. and the wound is closed without drainage. Re-exploration may or may not be indicated, depending on the patient s progress.

For a subdural abscess, adequate drainage may be accomplished

through bilateral trephine openings After the abscess is evacuated, the area is irrigated with a penicillin solution and the patient is given large doses of the antibiotic parenterally This treatment often suffices for cure Only occasionally is it necessary to instill the antibiotic solution into the area after the initial exploration When a subdural abscess does not respond to treatment, the reason may be that extensions of it are present in unusual positions, such as between the hemispheres In such cases, exposure of the purulent collection, either by an adequate craniotomy, or by introducing a brain cannula into the area of suppuration, is indicated Removal of the pus and injection of penicillin solution into the area effected a cure in 2 cases of an interhemispheric purulent collection in our series

An encapsulated brain abscess, provided it is free of retained foreign matter or bone fragments, can be evacuated by repeated aspiration, followed by instillation of penicillin (10,000 to 50,000 units in 1 cc aqueous solution). After the initial evacuation, the abscess cavity can be visualized by instilling 2 to 4 cc of Thorotrast, which stains the abscess wall Repeated roentgenography thereafter reveals the state of the lesion [41] Repeated aspiration may be necessary as the abscess outline enlarges In selected cases, excision of the abscess is indicated.

In the case of a cerebritis or abscess due to retained foreign matter or bone fragments, the inflamed area is excised completely, penicillin solution is instilled into the area, and the wound is closed tightly If there is extensive loss of scalp and bone, or if there is a fungus cerebri, the involved area is excised completely and covered by a temporary dural graft After granulations form, the dural graft is removed and skin is grafted upon the granulating surface (*see* Figs 92-93).

A fungus cerebri with a brain abscess, or a cerebritis, in an improperly debrided wound with a large opening in the dura, should be carefully evaluated by roentgenography before treatment is started. The area of involvement must be completely excised, mainly by suction If there is a heavy fibroglial scar, it may be removed with a sharp instrument If possible, a temporary dural graft should be used to close

the dural defect, especially if there is loss of scalp the goal being to make the dural sac a completely closed one If that is impossible, repeated lumbar punctures, as suggested by Browder[8, 16] or continuous lumber drainage may be used in order to prevent increased intracranial pressure and the risk of cerebral herniation Cerebral herniation or fungus cerebri in such cases is the result of a combination of infected cerebral tissue, an open dura, and usually a meningitis, in the absence of meningeal or cerebral infection an open dura would not result in herniation or fungus formation If the area can be covered by scalp, 10,000 to 50,000 units of penicillin in 1 cc. aqueous solution may be injected daily for 3 or 4 days. Some, however prefer to debride such wounds without closing the dura

If the patient fails to improve after an intracranial abscess has been evacuated, other foci may be present where they are not easily accessible to exploration. Nevertheless, an attempt must be made to locate the focus or foci of infection. Should a focus be found and completely evacuated, and adequate antibiotic therapy instituted, the patient may recover

Whatever the location of the abscess, a pneumoencephalogram is essential before the patient is discharged from the hospital. This is to make certain that no unsuspected collection of pus remains in the cranial cavity

Posttraumatic Epilepsy

Medical Management

Satisfactory control of posttraumatic epilepsy is obtained in most patients by the use of the anticonvulsant drugs, such as phenobarbital Dilantin Hydantal, Mesantoin and Mysoline Recurrence of seizures can usually be traced to dietary or other indiscretions or failure to use the drug The majority of patients can lead a normal life if they are careful in diet and in the use of medication if their home life is regular, and if their psychologic background is healthy An occasional patient

The preoperative preparation should include a detailed history, a complete neurologic examination, skull roentgenograms, and an electroencephalogram, the last-mentioned is particularly useful if it is obtained in association with metrazol activation, since this may help to localize the epileptogenic focus accurately Pneumoencephalograms are important, and in some cases angiograms are desirable Electrocorticography, which may reveal the epileptogenic focus, is best done under local anesthesia This may necessitate a two-stage operation The first stage consists of turning down a bone flap under general anesthesia, the second, 3 or 4 days later, of reopening the wound under local anesthesia, opening the dura, and proceeding with the electrocorticography and whatever excision is indicated However, we have now used a one-stage procedure in 8 consecutive cases, with satisfactory results, by means of local anesthesia and morphine intravenously for analgesia

Electrocorticography may reveal foci of abnormality elsewhere in the brain as well as in the intermediate zone which surrounds the scar proper, and which is the epileptogenic focus Foci 2 to 3 cm from the scar should be excised, those elsewhere should be left alone However, new foci may appear on the postoperative electrocorticogram (*see* Fig 72), demonstrating that excision of the scar and surrounding cortex may not suffice for cure Excision of the scar without electrocorticography in many cases has produced good clinical results, at least as good as those reported with the use of electrocorticography [24, 60]

In those cases in which the scar involves a greater portion of a lobe, the excision may have to be extended to the ventricle, or it may have to be extended in depth down to the white matter until all the scar tissue is excised The frontal or the occipital pole may be excised, if these structures are involved We use silver clips for hemostasis and the knife for excision Electrocoagulation is used sparingly. In all procedures, meticulous care to control hemorrhage and prevent infection is essential.

Postoperatively, the patient is given anticonvulsants, *e.g.*, phenobarbital and Dilantin, for several months or as long as may seem necessary.

A series of postoperative electroencephalograms may help in deciding on a therapeutic regimen

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